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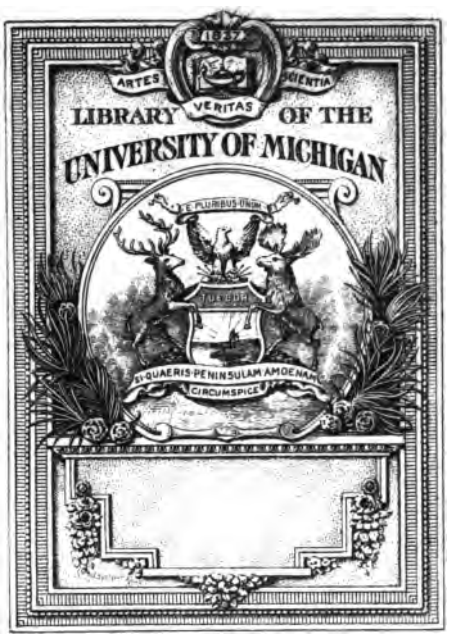
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FOR THE SESSION OF 1865-66.

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BALLARD, EDWARD, M.D.
BARLOW, GEORGE HILARO, M.D.
BRINTON, WILLIAM, M.D., F.R.S.
BROOKE, CHARLES, M.A., F.R.S.
BURROWS, GEORGE, M.D., F.R.S.
BUSK, GEORGE, F.R.S.
CLARK, FREDERICK LE GROS.
COCK, EDWARD.
CURLING, THOMAS BLIZARD, F.R.S.
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HEWETT, PRESCOTT GARDNER.
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1805. WILLIAM SAUNDERS, M.D.
1808. MATTHEW BAILLIE, M.D.
1810. SIR HENRY HALFORD, Bart., M.D., G.C.H.
1813. SIR GILBERT BLANE, Bart., M.D.
1815. HENRY CLINE.
1817. WILLIAM BABINGTON, M.D.
1819. SIR ASTLEY PASTON COOPER, Bart., K.C.H., D.C.L.
1821. JOHN COOKE, M.D.
1823. JOHN ABERNETHY.
1825. GEORGE BIRKBECK, M.D.
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1829. PETER MARK ROGET, M.D.
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1833. JOHN ELLIOTSON, M.D.
1835. HENRY EARLE.
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1839. SIR BENJAMIN COLLINS BRODIE, Bart., D.C.L.
1841. ROBERT WILLIAMS, M.D.
1843. EDWARD STANLEY.
1845. WILLIAM FREDERICK CHAMBERS, M.D., K.C.H.
1847. JAMES MONCRIEFF ARNOTT.
1849. THOMAS ADDISON, M.D.
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1857. SIR CHARLES LOCOCK, Bart., M.D.
1859. FREDERIC CARPENTER SKEY.
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OF THE

ROYAL MEDICAL AND CHIRURGICAL SOCIETY

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The figures succeeding the words *Trans.* and *Pro.* show the number of Papers which have been contributed to the Transactions or Proceedings by the Fellow to whose name they are annexed. *Sci. Com.* is attached to the names of those who have served on the Scientific Committees of the Society.

OCTOBER, 1865.

Those marked thus (†) have paid the Composition Fee in lieu of further annual subscriptions.

Amongst the non-residents, those marked thus (*) are entitled by composition to receive the Transactions.

Elected

1841 *Abercrombie, James, M.D., Cape of Good Hope.

1846 *Abercrombie, John, M.D., Physician to the Cheltenham General Hospital, 13, Suffolk square, Cheltenham.

1851 *Acland, Henry Wentworth, M.D., F.R.S., Honorary Physician to H.R.H. the Prince of Wales; Physician to the Radcliffe Infirmary; Regius Professor of Medicine, and Clinical Professor in the University of Oxford.

1847 Acosta, Elisha, M.D., New York, U.S.

1842 Acton, William, 17, Queen Anne street, Cavendish square. *Trans.* 1.

Elected

1851 ADAMS, JOHN, Surgeon to, and Lecturer on Descriptive and Surgical Anatomy at, the London Hospital; 10, Finsbury Circus. *Trans.* 3.

1852 ADAMS, WILLIAM, Surgeon to the Royal Orthopædic Hospital; 5, Henrietta street, Cavendish square. *Trans.* 2.

1837 *AINSWORTH, RALPH FAWSETT, M.D., Physician to the Manchester Royal Infirmary; Cliff Point, Lower Broughton, Manchester.

1839 ALCOCK, SIR RUTHERFORD, K.C.B., K.C.T., K.T.S., H.M.'s Envoy Extraordinary at the Court of Pekin. *Trans.* 1.

1826 ALDERSON, JAMES, M.D., F.R.S., *President*, Senior Physician to, and Lecturer on Clinical Medicine at, St. Mary's Hospital; 17, Berkeley square. S. 1829. C. 1848. T. 1849. V.P. 1852-3. P. 1865. *Trans.* 3.

1843 ALDIS, CHARLES JAMES BERRIDGE, M.D., Medical Officer of Health for St. George's, Hanover square; Senior Physician to the Surrey Dispensary; and Physician to the St. Paul and St. Barnabas Dispensary; 1, Chester terrace, Chester square. *Trans.* 2.

1850 ALEXANDER, CHARLES REVANS, Surgeon to the Royal Infirmary for Diseases of the Eye; 6, Cork street, Bond street.

1863 ALTHAUS, JULIUS, M.D., Physician to the Royal Infirmary for Diseases of the Chest; 18, Bryanston street, Portman square.

1862 ANDREW, EDWYN, M.D., Windsor House, Shrewsbury.

1862 ANDREW, JAMES, M.D., Assistant Physician to St. Bartholomew's Hospital; 59, Russell square.

1820 ANDREWS, THOMAS, M.D., Norfolk, Virginia.

1819 †ARNOTT, JAMES MONCRIEFF, F.R.S., Surgeon Extraordinary to H.M. the Queen. L. 1826-8. V.P. 1832-3. T. 1835-40. C. 1846, 1855-6. P. 1847-8. *Trans.* 8.

1817 †ASHBURNER, JOHN, M.D., F.L.S., 7, Hyde park place, Cumberland gate. C. 1821, 1830-1.

1851 ASHTON, THOMAS JOHN, Consulting Surgeon to the St. Marylebone Infirmary; 31, Cavendish square.

Elected

1825 †BABINGTON, BENJAMIN GUY, M.D., F.R.S., Physician to the Asylum for Deaf and Dumb; Consulting Physician to the German Hospital, and to the City of London Hospital for Diseases of the Chest; 31, George street, Hanover square. C. 1829. V.P. 1845-6. T. 1848. P. 1861-2. *Trans.* 2.

1820 *BADLEY, JOHN, Dudley, Worcestershire.

1840 BAINBRIDGE, WILLIAM, 47, Bridge street, Southwark.

1836 BAIRD, ANDREW WOOD, M.D., Physician to the Dover Hospital; Dover, Kent.

1851 *BAKER, ALFRED, Surgeon to the Birmingham General Hospital, and Lecturer on Surgery at Sydenham College; Cannon street, Birmingham.

1865 BAKER, WILLIAM MORRANT, Demonstrator of Anatomy, St. Bartholomew's Hospital; the College, St. Bartholomew's Hospital. *Trans.* 2.

1839 †BALFOUR, THOMAS GRAHAM, M.D., F.R.S., Deputy Inspector-General of Hospitals; 10, Sumner place, Onslow sq., Brompton. C. 1852-3. V.P. 1860-1. *Trans.* 2.

1848 BALLARD, EDWARD, M.D., Medical Officer of Health for Islington; 7, Compton terrace, Upper street, Islington. *Trans.* 2.

1849 BALLARD, THOMAS, M.D., 10, Southwick place, Hyde park.

1847 BARCLAY, ANDREW WHYTE, M.D., Physician to, and Lecturer on Materia Medica at, St. George's Hospital; Medical Officer of Health for Chelsea; 23A, Bruton street, Berkeley square. S. 1857-60. L. 1861-2. C. 1865. *Trans.* 2.

1848 BARKER, EDGAR, 9, Oxford square, Hyde park.

1862 BARKER, EDGAR, jun., late Surgeon to the Western General Dispensary; 5, Albion place, Hyde park square.

1833 †BARKER, THOMAS ALFRED, M.D., Senior Physician to, and Lecturer on Clinical Medicine at, St. Thomas's Hospital; 27, Wimpole street. C. 1844-5. V.P. 1853-4. T. 1860-2. *Trans.* 6.

1843 BARKER, THOMAS HERBERT, M.D., F.R.S.E., Senior Surgeon to the Bedford General Dispensary; Harpur place, Bedford.

Elected

1847 BARLOW, GEORGE HILARO, M.D., Physician to, and Lecturer on Clinical Medicine at, Guy's Hospital; Physician to the Magdalen Hospital; 12, Union street, Southwark. C. 1859.

1861 BARNES, ROBERT, M.D., Obstetric Physician to, and Lecturer on Midwifery at, St. Thomas's Hospital, and Physician to the Royal Maternity Charity; 46, Finsbury square. *Trans.* 3.

1864 BARRATT, JOSEPH GILLMAN, M.D., Physician to the London Surgical Home; 22, Cleveland gardens, Bayswater.

1840 BARROW, BENJAMIN, Surgeon to the Royal Isle of Wight Infirmary; Clifton House, Ryde, Isle of Wight.

1859 BARWELL, RICHARD, Assistant-Surgeon to, and Lecturer on Comparative Anatomy at, the Charing Cross Hospital; 32, George street, Hanover square. *Trans.* 1.

1844 BASHAM, WILLIAM RICHARD, M.D., *Vice-President*, Senior Physician to, and Lecturer on Medicine at, the Westminster Hospital; 17, Chester street, Grosvenor place. S. 1852-4. C. 1860-1. V.P. 1864-5. *Trans.* 2.

1862 BAZIRE, PIERRE VICTOR, M.D., Assistant-Physician to the National Hospital for the Paralysed and Epileptic; 28, Woburn square.

1862 BEALE, LIONEL SMITH, M.B., F.R.S., Professor of Physiology and General and Morbid Anatomy in King's College, London, and Physician to King's College Hospital; 61, Grosvenor street.

1860 BEALEY, ADAM, M.D., M.A. Camb., Physician to the Royal General Dispensary, St. Pancras; 27, Tavistock square.

1841 BEAMAN, GEORGE, M.D., 3, Henrietta street, Covent Garden.

1856 BEARDSLEY, AMOS, Bay villa grange, Newton in Cartmel, Lancashire.

1865 BEATTIE, HENRY, M.D., 37, Great Russell street, Bedford square.

1836 BEAUMONT, WILLIAM R., Consulting Surgeon to the Toronto General Hospital, late Professor of Surgery in the University of King's College; Toronto, Canada West. *Trans.* 3.

Elected

1840 BEEVOR, CHARLES, 41, Upper Harley street.

1858 BEGLEY, WILLIAM CHAPMAN, M.D., Middlesex County Lunatic Asylum, Hanwell.

1819 †BELL, THOMAS, F.R.S., F.L.S., The Wakes, Selborne, Hants. C. 1832-3. V.P. 1854. *Trans.* 1.

1847 BENNET, JAMES HENRY, M.D., The Ferns, Weybridge, and Mentone.

1845 BERRY, EDWARD UNWIN, 76, Gower street, Bedford square.

1820 BERTIN, STEPHEN, Paris.

1865 *BICKERSTETH, EDWARD ROBERT, Surgeon to the Royal Infirmary, Liverpool, and Lecturer on Clinical Surgery in the Liverpool School of Medicine; 2, Rodney street, Liverpool.

1815 †BILLING, ARCHIBALD, M.D., F.R.S., Member of the Senate of the University of London; 6, Grosvenor gate. C. 1825. V.P. 1828-9.

1827 BIRCH, WILLIAM, Barton-under-Needwood, Staffordshire. *Trans.* 2.

1855 BIRD, PETER HINCKES, F.L.S., 1, Norfolk square, Hyde park.

1856 BIRD, WILLIAM, Surgeon to the West London Hospital; 7, George street, Hanover square.

1849 BIRKETT, EDMUND LLOYD, M.D., Physician to the City of London Hospital for Diseases of the Chest; 48, Russell square. C. 1865.

1851 BIRKETT, GEORGE, M.D., Resident Proprietor, Northumberland House, Green Lanes, Stoke Newington.

1851 BIRKETT, JOHN, F.L.S., *Secretary*, Surgeon to, and Lecturer on Surgery at, Guy's Hospital; 59, Green street, Grosvenor square. L. 1856-7. S. 1863-5. *Trans.* 4. *Sci. Com.*

1846 BIRT, HUGH, British Naval Hospital, Valparaiso, Chili.

1843 BLACK, PATRICK, M.D., Physician to, and Lecturer on Medicine at, St. Bartholomew's Hospital; 11, Queen Anne street, Cavendish square. C. 1856.

1847 BLACKMAN, GEORGE C., M.D., Professor of Surgery in the Medical College of Ohio; New York, U.S.

1840 BLAKISTON, PEYTON, M.D., F.R.S., St. Leonard's-on-Sea.

Elected

1865 BLANCHET, HILARION, Examiner to the College of Physicians and Surgeons, Lower Canada; 6, Palace street, Quebec, Canada East.

1865 BLANDFORD, GEORGE FIELDING, M.B., 3, Clarges street, Piccadilly.

1845 BLENKINSOP, HENRY, Senior Surgeon to the Warwick Dispensary; Jury street, Warwick.

1823 BOJANUS, LOUIS HENRY, M.D., Wilna.

1846 BOSTOCK, JOHN ASHTON, Hon. Surgeon to H.M. the Queen; Surgeon-Major, Scots Fusilier Guards; 54, Chester square, Belgravia. C. 1861-2.

1863 BOWEN, FRANCIS, M.D., 62, Upper Berkeley street, Portman square.

1841 BOWMAN, WILLIAM, F.R.S., Surgeon to the Royal London Ophthalmic Hospital, Moorfields; 5, Clifford street, Bond street. C. 1852-3. V.P. 1862. *Trans.* 3.

1862 BRACE, WILLIAM HENRY, Surgeon to the Bath United Hospital; 1, Gay street, Bath.

1857 BRINTON, WILLIAM, M.D., F.R.S., 24, Brook street, Grosvenor square.

1851 BRODHURST, BERNARD EDWARD, Assistant-Surgeon to St. George's Hospital, and to the Royal Orthopædic Hospital; 20, Grosvenor street. *Trans.* 2; *Pro.* 1.

1844 †BROOKE, CHARLES, M.A., F.R.S., Surgeon to, and Lecturer on Surgery at, the Westminster Hospital; 16, Fitzroy square. C. 1855.

1854 *BROWN, HENRY, Surgeon to H.M. the Queen, and the Royal Household; Windsor.

1857 *BROWN, ROBERT, Surgeon to the Carlisle Dispensary; 4, Devonshire street, Carlisle.

1860 BROWN-SEQUARD, CHARLES EDOUARD, M.D., F.R.S., *Sci. Com.*

1851 BROWNE, ALEXANDER, M.D., Twynholm, Kirkcudbright.

1860 BRYANT, THOMAS, Assistant-Surgeon to, and Demonstrator of Operative Surgery at, Guy's Hospital; 2, Finsbury square. *Trans.* 4; *Pro.* 1. *Sci. Com.*

Elected

1855 BRYANT, WALTER JOHN, L.R.C.P. Edinb.; 7, Bathurst street, Hyde park gardens.

1823 BUCHANAN, B. BARTLET, M.D.

1864 BUCHANAN, GEORGE, M.D., Physician to the London Fever Hospital, and Assistant-Physician to the Hospital for Sick Children; Medical Inspector for the Privy Council; Medical Officer of Health for St. Giles District; 63, Harley street, Cavendish square.

1864 BUCKLE, FLEETWOOD, M.D., West Norfolk and Lynn Hospital; King's Lynn, Norfolk.

1839 BUDD, GEORGE, M.D., F.R.S., Consulting Physician to the Seamen's Hospital Ship, 'Dreadnought;' 20, Dover street, Piccadilly. C. 1846-7. V.P. 1857. *Trans.* 5.

1839 BURGESS, THOMAS HENRY, M.D.; Portsmouth.

1853 BURKE, PATRICK, 12, Upper Montagu street, Montagu square.

1833 †BURROWS, GEORGE, M.D., F.R.S., President of the Medical Council; Consulting Physician to St. Bartholomew's Hospital; Physician to Christ's Hospital; 18, Cavendish square. C. 1839-40, 1858-9. T. 1845-7. V.P. 1849-50. *Trans.* 2.

1820 BURROWS, SAMUEL.

1837 BUSK, GEORGE, F.R.S., F.L.S., Examiner in Comparative Anatomy at the University of London; Surgeon to the Seamen's Hospital Ship 'Dreadnought;' 15, Harley street, Cavendish square. C. 1847-8. V.P. 1855. *Trans.* 4.

1818 BUTTER, JOHN, M.D., F.R.S., F.L.S., Physician Extraordinary to the Plymouth Royal Eye Infirmary; Windsor villas, Plymouth.

1851 *CADGE, WILLIAM, Surgeon to the Norfolk and Norwich Hospital; 24, St. Giles's street, Norwich. *Trans.* 1.

1851 CALLAWAY, THOMAS, Algiers.

1861 CALLENDER, GEORGE WILLIAM, Assistant-Surgeon to, and Lecturer on Anatomy at, St. Bartholomew's Hospital; 47, Queen Anne street, Cavendish square. *Trans.* 1. *Sci. Com.*

Elected

1852 *CANNEY, GEORGE, M.D., Bishop-Auckland, Darlington, Durham.

1847 CARLILL, JOHN BURFORD, M.D., Surgeon-Accoucheur to the Newman street Lying-in Institution; 57, Berners street.

1853 CARTER, ROBERT BRUDENELL, Stroud, Gloucestershire.

1845 CARTWRIGHT, SAMUEL, Professor of Dental Surgery at King's College, London; Surgeon-Dentist to King's College Hospital; 32, Old Burlington street. C. 1860-1. *Sci. Com.*

1839 CATHROW, WILLIAM, 42, Weymouth street, Portland place. C. 1860-1.

1845 CHALK, WILLIAM OLIVER, Surgeon to the St. Marylebone Eye and Ear Institution; 3, Nottingham terrace, York gate, Regent's park.

1844 CHAMBERS, THOMAS KING, M.D., Hon. Physician to H.R.H. the Prince of Wales; Consulting Physician to, and Lecturer on Medicine at, St. Mary's Hospital; Consulting Physician to the Lock Hospital; 22B, Brook street, Grosvenor square. *Trans.* I. C. 1861.

1859 CHANCE, FRANK, M.D., Hastings.

1849 CHAPMAN, FREDERICK, Old Friars, Richmond green, Surrey.

1837 CHAPMAN, HENRY THOMAS, 16, Lower Seymour street, Portman square. C. 1858.

1852 CHILDS, GEORGE BORLASE, Surgeon-in-Chief to the City Police Force, and Surgeon to the Metropolitan Free Hospital; 11, Finsbury place south.

1865 CHOLMELEY, WILLIAM, M.D., Physician to the Great Northern Hospital; 40, Russell square.

1842 CHOWNE, WILLIAM DINGLE, M.D., Physician to, and Lecturer on Medicine and Midwifery at, the Charing Cross Hospital; Corresponding Fellow of the Royal Academy of Surgery of Madrid; 8, Connaught place West, Hyde park. C. 1853-4.

1860 CLARK, ANDREW, M.D., Assistant-Physician to, and Lecturer on Medicine at, the London Hospital; 23, Montague place, Russell square.

Elected

1839 †CLARK, FREDERICK LE GROS, Surgeon to, and Lecturer on Surgery at, St. Thomas's Hospital; Surgeon to the Magdalen Hospital; late Examiner in Surgery to the Royal College of Physicians; Consulting Surgeon to the Western General Dispensary, and to the London Female Penitentiary, Pentonville; 14, St. Thomas's street, Southwark, and Lee, Kent. S. 1847-9. V.P., 1855-6. *Trans.* 3.

1848 CLARKE, JOHN, M.D., Physician to the British Lying-in Hospital, and Physician to the General Lying-in Hospital; 42, Hertford street. May fair.

1861 *CLARKE. WILLIAM JAMES, Surgeon to the Huddersfield Infirmary; John William street, Huddersfield, Yorkshire.

1850 CLARKSON, JOSIAH, New Hall street, Birmingham. *Trans.* 1.

1842 CLAYTON, OSCAR MOORE PASSEY, 87, Harley street. C. 1865.

1853 CLOVER, JOSEPH THOMAS, 3, Cavendish place, Cavendish square.

1857 COATES, CHARLES, F.R.C.P. Edinb., Physician to the Bath United General Hospital; 10, Circus, Bath.

1851 COCK, EDWARD, Senior Surgeon to, and Lecturer on Clinical Surgery at, Guy's Hospital; Consulting Surgeon to the Asylum for Deaf and Dumb; Dean street, South, Tooley street, Southwark. C. 1857. *Trans.* 3.

1850 COHEN, DANIEL WHITAKER, M.D.; 26, Oxford rd., Kilburn.

1835 *COLBORNE, WILLIAM, Chippenham, Wiltshire.

1818 COLE, ROBERT, F.L.S., Holybourne, Hampshire.

1855 COLLINS, FREDERICK, M.D., Medical Officer of Health for Wanstead; Wanstead Lodge, Essex.

1828 CONOLLY, JOHN, M.D., D.C.L., Consulting Physician to the Middlesex County Lunatic Asylum, Hanwell.

1840 *COOKE, WILLIAM ROBERT, Osborne House, Lower Norwood.

1865 COOPER, ALFRED, Assistant-Surgeon to St. Mark's Hospital; 70, Jermyn street, Piccadilly.

1819 COOPER, GEORGE, Brentford, Middlesex.

Elected

1841 COOPER, GEORGE LEWIS, one of the Surgeons to the National Vaccine Institution, and Teacher of Vaccination to the Medical School of University College; Surgeon to the Bloomsbury Dispensary; 7, Woburn place, Russell square. C. 1860-1.

1843 COOPER, WILLIAM WHITE, Surgeon-Oculist in Ordinary to H.M. the Queen; and Hon. Consulting Ophthalmic Surgeon to St. Mary's Hospital; 19, Berkeley square. C. 1858-9.

1841 COOTE, HOLMES, Surgeon to, and Lecturer on Surgery at, St. Bartholomew's Hospital; 13, Queen Anne street, Cavendish square. S. 1853-4. C. 1864-5. *Trans.* 2.

1835 COPELAND, GEORGE FORD, Cheltenham.

1822 †COPLAND, JAMES, M.D., F.R.S., Consulting Physician to the Royal Infirmary for Children, and to the Great Northern Hospital, King's Cross; Hon. Fellow of the Royal Academy of Sciences of Sweden, &c.; 5, Old Burlington street. C. 1831. V.P. 1838-9. P. 1853-4.

1860 *CORRY, THOMAS CHARLES STEUART, M.D., Surgeon to the Belfast General Dispensary; 9, Clarendon place, Belfast.

1839 *CORSELLIS, CHARLES CÆSAR, M.D., F.L.S., Benson, Oxon.

1853 CORY, WILLIAM GILLETT, M.D.

1847 COTTON, RICHARD PAYNE, M.D., Physician to the Hospital for Consumption and Diseases of the Chest; 46, Clarges street, Piccadilly. C. 1863.

1828 †COULSON, WILLIAM, Hon. Consulting Surgeon to St. Mary's Hospital, and to the German Hospital; 2, Frederick place, Old Jewry, and 1, Chester terrace, Regent's park. C. 1831. L. 1832-7. V.P. 1851-2. *Trans.* 1.

1864 COULSON, WALTER JOHN, Surgeon to the Lock Hospital, and Surgical Registrar to St. Mary's Hospital; 29, St. James's place.

1860 †COUPER, JOHN, Assistant-Surgeon to, and Lecturer on Physiology at, the London Hospital; 33, Finsbury Circus.

1862 COWELL, GEORGE, Surgeon to the St. George's and St. James's Dispensary; 4, St. George's square, Pimlico.

Elected

1841 CRAWFORD, MERVYN ARCHDALL NOTT, M.D., Wiesbaden. C. 1853-4.

1847 CRITCHETT, GEORGE, Surgeon to the Royal London Ophthalmic Hospital, Moorfields; 75, Harley street, Cavendish square. C. 1865. *Trans.* 1.

1862 CROMPTON, SAMUEL, 17A, Princess street, Manchester.

1837 CROOKES, JOHN FARRAR, 5, Waterloo Crescent, Dover.

1860 CROSS, RICHARD, M.D., Queen street, Scarborough.

1849 *CROWFOOT, WILLIAM EDWARD, Beccles, Suffolk.

1851 CUMMING, JAMES CAMERON, M.D., 1, Cadogan place, Sloane street.

1865 CURGENVEN, J. BRENDON, 11, Craven hill gardens, Bayswater.

1846 CURLING, HENRY, Surgeon to the Margate Royal Sea-Bathing Infirmary, and the Ramsgate Seamen's Infirmary; Ramsgate, Kent.

1837 CURLING, THOMAS BLIZARD, F.R.S., Surgeon to the London Hospital; 39, Grosvenor street. S. 1845-6. C. 1850. T. 1854-7. V.P. 1859. *Trans.* 13. *Pro.* 1. *Sci. Com.*

1847 CURREY, JOHN EDMUND, M.D., Lismore, County Waterford.

1822 CUSACK, CHRISTOPHER JOHN, Chateau d'Eu, France.

1852 CUTLER, THOMAS, M.D., Acting Physician at the Spa Waters; Spa, Belgium

1836 *DANIEL, JAMES STOCK, Ramsgate, Kent.

1848 DAUBENY, HENRY.

1846 DAVIES, FREDERICK, M.D., 124, Gower street, Bedford sq.

1847 DAVIES, JOHN, M.D., Physician Extraordinary to the Hertford General Infirmary, and Visiting Physician to the Hadham Palace Lunatic Asylum, Hertford.

1853 DAVIES, ROBERT COKER NASH, Rye, Sussex.

1852 DAVIES, WILLIAM, M.D., 10, Gay street, Bath.

1852 DAVIS, JOHN HALL, M.D., Physician Accoucheur to, and Lecturer on Midwifery at, the Middlesex Hospital; Physician to the Royal Maternity Charity; and Consulting Physician-Accoucheur to the St. Pancras Infirmary; 11, Harley street, Cavendish square.

1818 DAWSON, JAMES, Wray Castle, Windermere.

Elected

1847 DAY, GEORGE EDWARD, M.D., F.R.S., Emeritus Professor of Medicine in the University of St. Andrew's; St. Mary Church, Torquay.

1858 DELIMA, TEOFILO, M.D., Caracas, Venezuela, South America.

1846 *DENTON, SAMUEL BEST, M.D., Ivy Lodge, Hornsea, Hull.

1859 DICKINSON, WILLIAM HOWSHIP, M.D., Curator of the Pathological Museum at St. George's Hospital; Assistant-Physician to the Hospital for Sick Children; 11, Chesterfield street, May fair. *Trans.* 6.

1844 DICKSON, ROBERT, M.D., F.L.S., Physician to the Scottish Hospital; 16, Hertford street, May fair. C. 1860.

1839 †DIXON, JAMES, Surgeon to the Royal London Ophthalmic Hospital, Moorfields; and Consulting Ophthalmic Surgeon to the Asylum for Idiots; 2, Portman square. L. 1849-55. V.P., 1857-8. T. 1863-4. *Trans.* 4.

1862 DOBELL, HORACE B., M.D., Physician to the Royal Infirmary for Diseases of the Chest, City road; 41, Harley street. *Trans.* 1.

1845 DODD, JOHN.

1857 DOUGLAS, ARCHIBALD, M.D., 8, Clifton place, Sussex square, Hyde park.

1863 DOWN, JOHN LANGDON HAYDON, M.D., Assistant-Physician to, and Lecturer on Materia Medica and Therapeutics at, the London Hospital; Physician to the Asylum for Idiots, Earlswood, Redhill. *Trans.* 1.

1853 DRUITT, ROBERT, M.R.C.P., Medical Officer of Health for St. George's, Hanover square; 37, Hertford street, May fair. *Trans.* 2.

1865 DRYSDALE, CHARLES ROBERT, M.D., Physician to the Farringdon Dispensary; 99, Southampton row, Russell square.

1845 DUFF, GEORGE, M.D., High street, Elgin.

1845 DUFFIN, EDWARD WILLSON, 18, Devonshire street, Portland place. *Trans.* 1.

1833 †DUNN, ROBERT, 31, Norfolk street, Strand. C. 1845-6. *Trans.* 2.

Elected

1861 Du Pasquier, Claudius Francis, Surgeon-Apothecary to H.M. the Queen, and to the Household of H.R.H. the Prince of Wales; 62, Pall Mall.

1863 Durham, Arthur Edward, Assistant-Surgeon to, and Lecturer on Anatomy at, Guy's Hospital; 30, Brook street, Grosvenor square. *Trans.* 2.

1843 Durrant, Christopher Mercer, M.D., Physician to the East Suffolk and Ipswich Hospital; Ipswich, Suffolk.

1839 Dyer, Henry Sumner, M.D., 37, Bryanston square. C. 1854-5.

1836 Earle, James William, late of Norwich.

1824 Edwards, George.

1823 Egerton, Charles Chandler, Kendall Lodge, Epping.

1861 *Elliot, Robert, M.D., Physician to the Carlisle Dispensary; 18, Lowther street, Carlisle.

1848 Ellis, George Viner, Examiner in Anatomy at the University of London; Professor of Anatomy in University College, London; University College, Gower street. C. 1863-4. *Trans.* 2.

1854 *Ellison, James, M.D., Surgeon in Ordinary to the Royal Household, Windsor; 14, High street, Windsor.

1835 England, William, M.D., Ipswich, Suffolk.

1842 Erichsen, John, Professor of Surgery in University College, London, and Surgeon to University College Hospital; Examiner in Surgery at the University of London; 6, Cavendish place, Cavendish square. C. 1855-6. *Trans.* 2.

1836 Evans, George Fabian, M.D., Physician to the General Hospital, Birmingham.

1815 *Evans, Griffith Francis Dorsett, M.D. C. 1838.

1845 Evans, William Julian, M.D., Pinner, Middlesex.

1864 Fagge, Charles Hilton, M.D., Demonstrator of Anatomy at Guy's Hospital; Physician to the Royal Infirmary for Diseases of Children and Women, Waterloo road; 43, Trinity square, Southwark.

1858 Falconer, Randle Wilbraham, M.D., Physician to the Bath United Hospital; 22, Bennett street, Bath.

Elected

1862 FARQUHARSON, ROBERT, M.D., Coldstream Guards' Hospital, Vincent square, Westminster.

1844 FARRE, ARTHUR, M.D., F.R.S., Physician-Accoucheur to H.R.H. the Princess of Wales and H.R.H. the Princess Louis of Hesse; 12, Hertford street, May fair. C. 1857. V.P. 1864. *Sci. Com.*

1863 FENWICK, SAMUEL, M.D., Assistant-Physician to the City of London Hospital for Diseases of the Chest; 74, Harley street, Cavendish square. *Trans.* 2.

1841 FERGUSSON, WILLIAM, F.R.S., Surgeon Extraordinary to H.M. the Queen; Professor of Surgery in King's College, London, and Surgeon to King's College Hospital; Professor of Anatomy and Surgery at the Royal College of Surgeons; 16, George street, Hanover square. C. 1849-50. V.P. 1863-4. *Trans.* 4.

1852 *FIELD, ALFRED GEORGE, Surgeon to St. Mary's Hospital, Brighton; 28, Old Steine, Brighton.

1849 FINCHAM, GEORGE TUPMAN, M.D., Physician to, and Lecturer on Clinical Medicine at, the Westminster Hospital; 2, Eccleston terrace south, Eccleston square.

1836 FISHER, SIR JOHN WILLIAM, late Surgeon in Chief to the Metropolitan Police Force; 5, Grosvenor gate. C. 1843-4.

1860 FITZGERALD, THOMAS GEORGE, Staff-Surgeon; 6, Whitehall yard.

1842 FLETCHER, THOMAS BELL ELCOCK, M.D., Physician to the Birmingham General Hospital; Waterloo street, Birmingham. *Trans.* 1.

1859 FLOWER, WILLIAM HENRY, F.R.S., Conservator of the Museum at the Royal College of Surgeons of England. *Trans.* 1.

1864 *FOLKER, WILLIAM HENRY, Surgeon to the North Staffordshire Infirmary, Hanley, Stoke-on-Trent, Staffordshire.

1848 FORBES, JOHN GREGORY, Surgeon to the Metropolitan Convalescent Institution; 9, Devonport street, Hyde park. *Trans.* 2.

Elected

1852 †FORSTER, JOHN COOPER, Assistant-Surgeon to, and Lecturer on Anatomy at, Guy's Hospital; Surgeon to the Royal Infirmary for Children, &c.; 10, St. Thomas's street, Southwark. *Pro.* 1.

1865 FOSTER, BALTHAZAR WALTER, M.D., Professor of Anatomy at the Queen's College, Birmingham, and Assistant-Physician to the Queen's Hospital; 55, Calthorpe street, Edgbaston, Birmingham.

1859 FOX, EDWARD LONG, M.B., Physician to the Bristol Royal Infirmary; 1, Chesterfield place, Clifton.

1858 *FOX, WILSON, M.D., Professor of Pathological Anatomy at University College, London, and Assistant-Physician to University College Hospital; 24, Henrietta street, Cavendish square. *Trans.* 2.

1841 FRANZ, JOHN CHRISTOPHER AUGUSTUS, M.D.

1843 FRASER, PATRICK, M.D., Physician to the London Hospital, and to the London Dispensary; 63, Grosvenor street.

1836 †FRENCH, JOHN GEORGE, Surgeon to the St. James's Infirmary; 41, Great Marlborough street. C. 1852-3.

1849 FREERE, ROBERT TEMPLE, M.A., F.R.C.P., 34, Upper Harley street.

1846 FULLER, HENRY WILLIAM, M.D., *Secretary*, Physician to St. George's Hospital; 13, Manchester square. C. 1862. S. 1864. *Trans.* 2.

1864 *GAIRDNER, WILLIAM TENNANT, M.D., Professor of the Practice of Medicine in the University of Glasgow; Physician to the Glasgow Royal Infirmary; 21, Blythswood square, Glasgow.

1860 GALTON, ROBERT CAMERON, M.D., Hadzor House, Droitwich.

1865 GANT, FREDERICK JAMES, Surgeon and Pathological Anatomist to the Royal Free Hospital; 5, Grenville street, Brunswick square, W.C.

1854 GARROD, ALFRED BARING, M.D., F.R.S., Professor of Materia Medica in King's College, London, and Physician to King's College Hospital; 84, Harley street, Cavendish square. *Trans.* 8.

Elected

1857 GASCOYEN, GEORGE GREEN, Surgeon to the Lock Hospital; Assistant-Surgeon to, and Lecturer on Descriptive and Surgical Anatomy at, St. Mary's Hospital; 48, Queen Anne street, Cavendish square. *Trans.* 1. *Sci. Com.*

1851 GASKOIN, GEORGE, 3, Westbourne park.

1819 GAULTER, HENRY.

1848 GAY, JOHN, Senior Surgeon to the Great Northern Hospital, and Consulting Surgeon to the Asylum for Idiots; 10, Finsbury place south.

1821 *GEORGE, RICHARD FRANCIS, late Senior Surgeon to the Bath General Hospital; 20, Marlborough buildings, Bath.

1864 GIBB, GEORGE DUNCAN, M.D., M.A., Assistant-Physician to, and Lecturer on Forensic Medicine at, the Westminster Hospital; 19A, Portman street, Portman sq. *Trans.* 1.

1858 GODFREY, BENJAMIN, M.D., Carlton House, Enfield, Middlesex.

1851 GOODFELLOW, STEPHEN JENNINGS, M.D., Physician to, and Lecturer on Medicine at, the Middlesex Hospital; 5, Savile row, Burlington gardens. C. 1864-5. *Trans.* 2.

1818 GORDON, JAMES ALEXANDER, M.D., C.B., F.R.S., Pixholme, near Dorking, Surrey. C. 1828. V.P. 1829. *Trans.* 1.

1862 GOULSTONE, JOHN G., M.D., Bellevue, Shrewsbury.

1851 GOWLLAND, PETER YEAMES, Surgeon to St. Mark's Hospital; 34, Finsbury square.

1844 GRANTHAM, JOHN, Crayford, Kent.

1846 GREAM, GEORGE THOMPSON, M.D., Physician-Accoucheur to H.R.H. the Princess of Wales; 2, Upper Brook street, Grosvenor square. C. 1863.

1843 GREENHALGH, ROBERT, M.D., Physician-Accoucheur to, and Lecturer on Midwifery at, St. Bartholomew's Hospital, and Consulting Physician to the Samaritan Free Hospital for Women and Children, and to the City of London Lying-in Hospital; 76, Grosvenor street.

1860 GREENHOW, EDWARD HEADLAM, M.D., Assistant-Physician to, and Lecturer on Public Health and on Medical Jurisprudence at, the Middlesex Hospital, and Consulting Physician to the Western General Dispensary; 77, Upper Berkeley street, Portman square. *Trans.* 2.

Elected

1814 GROVE, JOHN, M.D., Salisbury.

1852 GROVE, JOHN, West Hill, Wandsworth, Surrey.

1860 GUENEAU DE MUSSY, HENRY, M.D., 4, Cavendish place, Cavendish square.

1849 GULL, WILLIAM WITHEY, M.D., Physician to Guy's Hospital; Member of the Senate of the University of London; 26, Brook street, Grosvenor square. C. 1864. *Trans.* 2.

1837 GULLY, JAMES MANBY, M.D.; Great Malvern, Worcestershire.

1854 HABERSHON, SAMUEL OSBORNE, M.D., Assistant-Physician to, and Lecturer on Materia Medica and Therapeutics at, Guy's Hospital; Examiner in Materia Medica at the University of London; 22, Wimpole street, Cavendish square. *Trans.* 2.

1849 HAILEY, HAMMETT, Newport Pagnell, Bucks.

1845 HALL, SIR JOHN, M.D., K.C.B., Inspector-General of Hospitals; 39, Necker Strasse, Stuttgart.

1848 HALLEY, ALEXANDER, M.D., F.G.S., 7, Harley street, Cavendish square.

1819 †HAMMERTON, THOMAS, 112, Piccadilly. C. 1829-30.

1838 HANCOCK, HENRY, Surgeon to, and Lecturer on Surgery at, the Charing Cross Hospital; Surgeon to the Royal Westminster Ophthalmic Hospital; 37, Harley street, Cavendish square. C. 1851.

1849 *HANSARD, RICHARD JAMES, late Surgeon to the Radcliffe Infirmary, Oxford.

1848 *HARCOURT, GEORGE, M.D., Chertsey, Surrey.

1836 HARDING, JOHN FOSSE, Mount Sandford, Southborough, Tunbridge Wells. C. 1858-9.

1856 HARE, CHARLES JOHN, M.D., Professor of Clinical Medicine at University College, London; and Physician to University College Hospital; 41, Brook street, Grosvenor square.

1857 HARLEY, GEORGE, M.D., F.R.S., Professor of Medical Jurisprudence in University College, London; and Assistant-Physician to University College Hospital; 77, Harley street, Cavendish square. *Trans.* 1. *Sci. Com.* 2.

Elected

1864 HARLEY, JOHN, M.D., F.L.S., 78, Upper Berkeley street, Portman square.

1859 HARRIS, FRANCIS, M.D., Assistant-Physician to, and Lecturer on Botany at, St. Bartholomew's Hospital; Assistant-Physician to the Hospital for Sick Children; 24, Cavendish square.

1841 HARVEY, WILLIAM, Surgeon to the Royal Dispensary for Diseases of the Ear, and to the Freemasons' Female Charity, and Aural Surgeon to the Great Northern Hospital; 2, Soho square. C. 1854.

1855 HAVILAND, ALFRED, Surgeon to the Bridgewater Infirmary; Bridgewater, Somerset.

1828 †HAWKINS, CÆSAR HENRY, F.R.S., Sergeant-Surgeon to H.M. the Queen, and Consulting Surgeon to St. George's Hospital; 26, Grosvenor street. C. 1830-1, 1860. V.P. 1838-9. T. 1841-4. P. 1855-6. *Trans.* 12.

1838 †HAWKINS, CHARLES, Inspector of Anatomical Schools in London; Consulting Surgeon to Queen Charlotte's Hospital; 22, Savile row, Burlington gardens. C. 1846-7. S. 1850. V.P. 1858. T. 1861-2. *Trans.* 2.

1848 HAWKSLEY, THOMAS, M.D., Physician to the Margaret street Dispensary for Consumption and Diseases of the Chest; 70, Brook street, Hanover square.

1860 HAYWARD, HENRY HOWARD, Assistant-Dental Surgeon to the Dental Hospital of London; Dental Surgeon to the Hospital for Consumption, Brompton; 18, Harley street, Cavendish square.

1861 HAYWARD, WILLIAM HENRY, Church House, Oldbury, near Birmingham.

1848 *HEALE, JAMES NEWTON, M.D., Physician to the Winchester County Hospital; Winchester, Hants.

1850 HEATON, GEORGE, M.D., Boston, U.S.

1829 †HEBERDEN, THOMAS, M.D., 43, Park street, Grosvenor square.

1844 HENNEN, JOHN, M.D. L. 1848-50.

1849 HENRIQUES, AMOS, M.D., Hon. Physician to the Spanish Embassy; 67, Upper Berkeley street, Portman square.

Elected

1821 HERBERSKI, VINCENT, M.D., Professor of Medicine in the University of Wilna.

1843 HEWETT, PRESCOTT GARDNER, Surgeon to St. George's Hospital; 1, Chesterfield street, May fair. C. 1859. *Trans.* 7. *Sci. Com.*

1855 HEWITT, GRAILY, M.D., Professor of Midwifery in University College, London, and Obstetric Physician to University College Hospital; Physician to the British Lying-in Hospital; 36, Berkeley square.

1853 HEWLETT, THOMAS, Surgeon to Harrow School; Harrow, Middlesex. *Trans.* 1.

1841 *HIGHMORE, NATHANIEL, Sherborne, Dorsetshire.

1862 HILL, MATTHEW BERKELEY, M.B. Lond., Assistant-Surgeon to University College Hospital; 14, Weymouth street, Portland place.

1854 HILLIER, THOMAS, M.D., Physician to the Hospital for Sick Children; Physician to the Skin Department of University College Hospital, and Medical Officer of Health for St. Pancras; 32, Queen Anne street, Cavendish square. *Trans.* 1.

1842 HILLMAN, WILLIAM AUGUSTUS, Senior Assistant-Surgeon to the Westminster Hospital; 1, Argyll street, Regent street. C. 1858-9.

1841 †HILTON, JOHN, F.R.S., Surgeon to Guy's Hospital; Consulting Surgeon to the Royal General Dispensary, St. Pancras; Examiner in Surgery at the University of London; 10, New Broad street, City. C. 1851. V.P. 1863-4. *Trans.* 3.

1859 HIRD, FRANCIS, Assistant-Surgeon to, and Lecturer at, the Charing Cross Hospital; 13, Old Burlington street.

1840 HODGKIN, THOMAS, M.D., Consulting Physician to the Hospital for Diseases of the Skin, and Member of the Senate of the University of London; 35, Bedford square. C. 1842-3. V.P. 1862-3. *Trans.* 6.

1813 HODGSON, JOSEPH, F.R.S., 60, Westbourne terrace, Hyde park gardens. C. 1817. P. 1851-2. *Trans.* 1.

1861 *HOFFMEISTER, WILLIAM CARTER, M.D., Surgeon to H.M. the Queen in the Isle of Wight; Cowes, Isle of Wight.

Elected

1843 HOLDEN, LUTHER, *Librarian*, Surgeon to, and Lecturer on Anatomy at, St. Bartholomew's Hospital; Surgeon to the Metropolitan Dispensary; Surgeon to the Foundling Hospital; 65, Gower street, Bedford square. C. 1859. L. 1865.

1814 †HOLLAND, SIR HENRY, Bart., M.D., D.C.L., LL.D., F.R.S., Physician in Ordinary to H.M. the Queen; 25, Brook street, Grosvenor square. C. 1817, 1833-4. V.P. 1826, 1840. *Trans.* 1.

1861 HOLMAN, WILLIAM HENRY, M.B. Lond.; 68, Adelaide road south, Hampstead.

1856 HOLMES, TIMOTHY, Assistant-Surgeon to, and Lecturer on Anatomy at, St. George's Hospital, and Surgeon to the Hospital for Sick Children; Surgeon in Chief to the Metropolitan Police Force; 22, Queen street, May fair. *Trans.* 2.

1846 HOLT, BARNARD WIGHT, Senior Surgeon to, and Lecturer on Clinical Surgery at, the Westminster Hospital; Medical Officer of Health for Westminster; 14, Savile row, Burlington gardens. C. 1862-3.

1846 HOLTHOUSE, CARSTEN, Surgeon to, and Lecturer on Surgery at, the Westminster Hospital; Surgeon to the South London Ophthalmic Hospital; 2, Storey's gate, St. James's park. C. 1863.

1853 HOOD, WILLIAM CHARLES, M.D., F.L.S., Visiting Physician in Lunacy to the Court of Chancery; Croydon Lodge, Surrey. *Trans.* 1.

1865 HOWARD, EDWARD, M.D., Redhill, Surrey.

1828 *HOWELL, EDWARD, M.D., Senior Consulting Physician to the Swansea Infirmary; 2, South Hill place, Swansea, Glamorganshire.

1857 HULKE, JOHN WHITAKER, Assistant-Surgeon to, and Lecturer on Ophthalmic Surgery at, the Middlesex Hospital, and Assistant-Surgeon to the Royal London Ophthalmic Hospital, Moorfields; 10, Old Burlington street. *Trans.* 4.

Elected

1857 HULME, EDWARD CHARLES, Surgeon to the Great Northern Hospital; Surgeon to the Central London Ophthalmic Hospital; 38, Gower street, Bedford square. *Trans.* 1.

1844 HUMBY, EDWIN, 83, Hamilton terrace, St. John's wood.

1855 HUMPHRY, GEORGE MURRAY, M.D., F.R.S., Surgeon to Addenbrooke's Hospital, and Lecturer on Anatomy in the Cambridge University Medical School; Cambridge. *Trans.* 4.

1849 HUSSEY, EDWARD LAW, Senior Surgeon to the Radcliffe Infirmary, and Consulting Surgeon to the County Lunatic Asylum and the Warneford Asylum; 104, St. Aldate's, Oxford. *Trans.* 1.

1856 HUTCHINSON, JONATHAN, Surgeon to, and Lecturer on Surgery at, the London Hospital; Assistant-Surgeon to the Royal London Ophthalmic Hospital, Moorfields; Surgeon to the Metropolitan Free Hospital; 4, Finsbury circus. *Pro.* 2.

1820 HUTCHINSON, WILLIAM, M.D.

1840 HUTTON, CHARLES, M.D., Senior Physician to the General Lying-in Hospital; 26, Lowndes street, Belgrave square. C. 1858-9.

1847 IMAGE, WILLIAM EDMUND, Senior Surgeon to the Suffolk General Hospital; Bury St. Edmund's, Suffolk. *Trans.* 1.

1856 INGLIS, CORNELIUS, M.D.; 2, Norfolk rd., St. John's wood.

1826 INGRAM, WILLIAM, Midhurst, Sussex.

1845 *JACKSON, HENRY, Senior Surgeon to the Sheffield General Infirmary; St. James's row, Sheffield, Yorkshire.

1841 JACKSON, PAUL, 24, Wimpole street, Cavendish square. C. 1862.

1863 JACKSON, THOMAS VINCENT, Surgeon to the South Staffordshire General Hospital; Darlington street, Wolverhampton.

1841 JACOBOVICS, MAXIMILIEN MORRIS, M.D., Vienna.

1825 JAMES, JOHN B., M.D.

1844 JEAFFRESON, SAMUEL JOHN, M.D., Physician to the Warneford Hospital, and Warwick Dispensary; Leamington, Warwickshire.

Elected

1839 JEFFREYS, JULIUS, F.R.S., Drymona, Belvidere road south, Upper Norwood, Surrey.

1840 *JENKS, GEORGE SAMUEL, M.D., 18, Circus, Bath.

1851 JENNER, WILLIAM, M.D., F.R.S., Physician in Ordinary to H.M. the Queen, and to H.R.H. the Prince of Wales; Physician to University College Hospital, and Professor of the Principles and Practice of Medicine at University College; 8, Harley street, Cavendish square. C. 1864. *Trans.* 3.

1848 JOHNSON, ATHOL ARCHIBALD WOOD, 20, Regency square, Brighton. *Trans.* 1.

1851 JOHNSON, EDMUND CHARLES, M.D., Corresponding Member of the Medical and Philosophical Society of Florence and of "L'Institut Genevois;" C., No. 3, Albany, Piccadilly.

1847 JOHNSON, GEORGE, M.D., Professor of the Principles and Practice of Medicine in King's College, London, and Physician to King's College Hospital; Member of the Senate of the University of London; 11, Savile row, Burlington gardens. C. 1862-3. *Trans.* 5.

1862 JONES, CHARLES HANDFIELD, M.B., F.R.S., Physician to, and Lecturer on Medicine at, St. Mary's Hospital; 49, Green street, Grosvenor square.

1844 †JONES, HENRY BENCE, M.A., M.D., F.R.S.; 31, Brook street, Grosvenor square. C. 1855-6. *Trans.* 11.

1835 JONES, HENRY DERVICHE, 12, Norfolk crescent, Oxford square. C. 1854-5.

1837 JONES, THOMAS WILLIAM, M.D., 19, Finsbury pavement, and Green street, Ponders End. C. 1858.

1859 JONES, WILLIAM PRICE, M.D., Surbiton, Kingston.

1865 JORDAN, FURNEAUX, Surgeon to the Queen's Hospital, and Senior Surgeon to the Birmingham Eye and Ear Hospital; 16, Colmore row, Birmingham.

1829 *JULIUS, GEORGE CHARLES, Richmond, Surrey.

1816 *KAUFFMAN, GEORGE HERMANN, M.D., Hanover.

1848 *KENDELL, DANIEL BURTON, M.D., Kettlethorpe Hall, Wakefield, Yorkshire.

1847 KEYSER, ALFRED, 21, Norfolk crescent, Oxford square.

Elected

1857 KIALLMARK, HENRY WALTER, 46, Prince's sq., Bayswater.

1851 KINGDON, JOHN ABERNETHY, Surgeon to the City of London Truss Society, and to the City Dispensary; 2, New Bank buildings, City. *Trans.* 1.

1855 LANE, JAMES ROBERT, Surgeon to, and Lecturer on Operative Surgery at, St. Mary's Hospital, and Surgeon to the Lock and St. Mark's Hospitals; 2, Berkeley street, Piccadilly.

1840 LANE, SAMUEL ARMSTRONG, *Vice President*, Surgeon to, and Lecturer on Surgery at, St. Mary's Hospital, and Consulting Surgeon to the Lock Hospital; 2, Berkeley street, Piccadilly. C. 1849-50. V.P. 1865.

1865 LANGTON, JOHN, Assistant Demonstrator at St. Bartholomew's Hospital; Assistant-Surgeon to the City of London Truss Society; the College, St. Bartholomew's Hospital.

1841 *LASHMAR, CHARLES, M.D., 83, North End, Croydon, Surrey.

1862 LATHAM, PETER WALLWORK, M.A., M.B., Physician to Addenbrooke's Hospital, Cambridge; Examiner for Medical Degrees in Cambridge University; 15, Sidney street, Cambridge.

1816 LAWRENCE, G. E.

1809 †LAWRENCE, WILLIAM, F.R.S., Sergeant-Surgeon to H.M. the Queen; Consulting Surgeon to St. Bartholomew's Hospital; Surgeon to Bridewell and Bethlem Hospital; Foreign Associate of the Imperial Academy of Medicine of Paris; 18, Whitehall place. S. 1813-17. V.P. 1818-19. T. 1821-6. P. 1831-2. C. 1820, 1833-4, 1842-3. *Trans.* 18.

1840 LAYCOCK, THOMAS, M.D., F.R.S.E., Professor of the Practice of Medicine and of Clinical Medicine, and Lecturer on Psychology and Mental Diseases in the University of Edinburgh, and Physician to the Edinburgh Royal Infirmary; 4, Rutland street, Edinburgh.

1843 *LEACH, JESSE, Moss Hall, Heywood, near Bury, Lancashire.

Elected

1822 LEDSAM, JOHN JOSEPH, M.D., 17, Esplanade, Scarborough, Yorkshire.

1823 †LEE, HENRY, M.D.. Weather Oak, Alvechurch, near Bromsgrove, Worcestershire. C. 1837. S. 1839-40.

1843 LEE, HENRY, Surgeon to, and Lecturer on Pathology at, St. George's Hospital; 9, Savile row, Burlington gardens. C. 1856-7. L. 1863-4. *Trans.* 8. *Pro.* 1.

1822 †LEE, ROBERT, M.D., F.R.S., Lecturer on Midwifery at St. George's Hospital; Corresponding Member of the Imperial Academy of Medicine, Paris; 4, Savile row, Burlington gardens. C. 1829, 1834. S. 1830-3. V.P. 1835. *Trans.* 27.

1836 LEIGHTON, FREDERICK, M.D., Frankfort-on-the-Maine.

1854 LEON, HANANEL DE, M.D., 15, Holland villas road, North Kensington.

1806 LIND, JOHN, M.D.

1819 LLOYD, ROBERT, M.D.

1820 LOCHER, J. G., M.C.D., Town Physician of Zurich. *Trans.* 2.

1824 †LOCOCK, SIR CHARLES, Bart., M.D., F.R.S., First Physician-Accoucheur to H.M. the Queen; Member of the Senate of the University of London; 26, Hertford street, Mayfair. C. 1826. V.P. 1841. P. 1857-8. *Trans.* 1.

1852 LODGE, CHARLES, M.D., "United States Army."

1846 LOMAX, HENRY THOMAS, Surgeon to the County Police; St. Mary's grove, Stafford.

1860 LONGMORE, THOMAS, Deputy Inspector-General and Professor of Clinical and Military Surgery, New Army Medical School, Royal Victoria Hospital, Netley, Southampton. *Trans.* 2.

1836 LÖWENFELD, JOSEPH S., M.D., Berbice.

1852 LUKE, JAMES, F.R.S., Consulting Surgeon to the London Hospital, and Surgeon to St. Luke's Hospital for Lunatics; Woolley Lodge, Maidenhead Thicket, Berks. C. 1858. *Trans.* 4.

1857 LYON, FELIX WILLIAM, M.D., Littlecote, Hungerford, Hants.

Elected

1862 *M'DONNELL, ROBERT, M.D., F.R.S., Lecturer on Surgery in the Carmichael School of Medicine, and Surgeon to Jervis street Hospital; 14, Lower Pembroke st., Dublin,

1846 M'EWEN, WILLIAM, M.D., Surgeon to Chester Castle; 27, Nicholas street, Chester.

1823 †MACILWAIN, GEORGE, Consulting Surgeon to the Finsbury Dispensary and the St. Ann's Society's Schools; 3, the Court yard, Albany. C. 1829-30. V.P. 1848. *Trans.* 1.

1822 MACINTOSH, RICHARD, M.D.

1859 *M'INTYRE, JOHN, M.D., Odiham, Hants.

1818 MACKENZIE, WILLIAM, M.D., Surgeon-Oculist to H.M. the Queen in Scotland, and Surgeon to the Glasgow Eye Infirmary; 49, Bath street, Glasgow. *Trans.* 2.

1834 *MACKINDER, DRAPER, M.D., Consulting Surgeon to the Dispensary, Gainsborough, Lincolnshire.

1844 MACLACHLAN, DANIEL, M.D., late Physician to the Royal Hospital, Chelsea; Deputy Inspector-General of Hospitals; Ventnor, Isle of Wight. C. 1860-1. Trans. 1.

1860 MACLEAN, JOHN, M.D., 24, Portman street, Portman square.

1849 MACLURE, DUNCAN MACLACHLAN, 16, Harley street, Cavendish square.

1842 MACNAUGHT, JOHN, M.D., Bedford street, Liverpool.

1837 M'WHINNIE, ANDREW MELVILLE, 7, Serjeant's Inn, Temple. C. 1851-2. *Trans.* 1.

1855 MARCET, WILLIAM, M.D., F.R.S., 1, Torrington street, Torrington square. *Trans.* 1. *Sci. Com.*

1848 MARKHAM, WILLIAM ORLANDO, M.D., Physician to St. Mary's Hospital; 3, Harley street. C. 1862-3. *Trans.* 2.

1838 MARSH, THOMAS PARR, M.D., Consulting Physician to the Salop Infirmary, Shrewsbury; Coed Cefer, Monmouth.

1851 MARSHALL, JOHN, F.R.S., Surgeon to University College Hospital; Fullerian Professor of Physiology at the Royal Institution of Great Britain; 10, Savile row, Burlington gardens. *Trans.* 2.

Elected

1841 MARTIN, SIR JAMES RANALD, C.B., F.R.S., Examining Medical Officer to the Secretary of State for India in Council, President of Medical Board for Examination of Officers of H.M.'s Indian Medical Service; Inspector General of Hospitals; 37, Upper Brook street. C. 1853. V.P. 1862.

1853 MASFEN, WILLIAM EDWARD, Surgeon to the Staffordshire General Infirmary; Stafford.

1864 MASON, FRANCIS, 10, Conduit street, Regent street.

1837 MAYO, THOMAS, M.D., F.R.S.; the Refuge, Yarmouth, Isle of Wight. S. 1841. C. 1847-8. V.P. 1851-2.

1839 MEADE, RICHARD HENRY, Senior Surgeon to the Bradford Infirmary; Bradford, Yorkshire. *Trans.* 1.

1865 MEDWIN, AARON GEORGE, M.D., 4, Blackheath road, Kent.

1837 MERRIMAN, SAMUEL WILLIAM JOHN, M.D., Consulting Physician-Accoucheur to the Westminster General Dispensary, and Physician-Accoucheur to the Western General Dispensary; 13, Godolphin road, New road, Hammersmith. C. 1851-2. *Trans.* 1.

1852 MERRYWEATHER, JAMES, Consulting Surgeon to the National Dental Hospital; 57, Brook street, Grosvenor square.

1847 MERYON, EDWARD, M.D., F.G.S., 14, Clarges street, Piccadilly. L. 1859-60. C. 1864-5. *Trans.* 1.

1815 MEYER, AUGUSTUS, M.D., St. Petersburgh.

1840 MIDDLEMORE, RICHARD, Consulting Surgeon to the Birmingham Eye Infirmary; Temple row, Birmingham.

1854 MIDDLESHIP, EDWARD ARCHIBALD, late of Richmond, Surrey.

1860 *MILES, HERBERT CHALMERS, Assistant-Surgeon in the Royal Artillery, Woolwich.

1818 *MILLER, PATRICK, M.D., F.R.S.E., Senior Physician to the Devon and Exeter Hospital, and to St. Thomas's Hospital for Lunatics; the Grove, Exeter, Devonshire.

1863 MONRO, HENRY, M.D., Physician to St. Luke's Hospital; 13, Cavendish square.

1844 MONTEFIORE, NATHANIEL, 36, Hyde park gardens.

Elected

1848 MOORE, CHARLES HEWITT, Surgeon to, and Lecturer on Anatomy at, the Middlesex Hospital; 102, Piccadilly. L. 1858. S. 1859-62. C. 1864-5. *Trans.* 7. *Sci. Com.*

1836 MOORE, GEORGE, M.D., Hastings, Sussex.

1861 MOREHEAD, CHARLES, M.D, Hon. Surgeon to H.M. the Queen; Deputy-Inspector General of Hospitals; late Principal of Grant Medical College, Bombay, &c.

1857 MORGAN, JOHN, 3, Sussex place, Hyde park gardens. *Trans.* 1.

1861 MORGAN, JOHN EDWARD, M.B., Lecturer on Pathology at the Manchester Royal School of Medicine; 3, Gore street, Piccadilly, Manchester.

1851 MOUAT, FREDERIC JOHN, M.D., Professor of Medicine in the Medical College of Calcutta, Secretary of the Council of Education in India, and Inspector-General of Gaols, Lower Provinces; Calcutta.

1856 MURCHISON, CHARLES, M.D., Senior Physician to the London Fever Hospital; Assistant-Physician to, and Lecturer on Pathological Anatomy at, the Middlesex Hospital; 79, Wimpole street, Cavendish square. *Trans.* 3.

1847 MURCHISON, SIMON, Bicester, Oxon.

1863 MYERS, ARTHUR B. R., Coldstream Guards' Hospital, Vincent square, Westminster.

1859 NAYLER, GEORGE, Assistant-Surgeon to the Royal Orthopædic Hospital; 8, George street, Hanover square.

1835 NELSON, THOMAS ANDREW, M.D., 10, Nottingham terrace, York gate, Regent's park.

1843 NEWTON, EDWARD, 30, Fitzroy square. C. 1863-4.

1849 NORMAN, HENRY BURFORD, Portland Lodge, Southsea, Hants.

1845 NORRIS, HENRY, Charmouth, Dorset.

1847 *NOURSE, WILLIAM EDWARD CHARLES, 11, Marlborough place, Brighton.

1849 NOVERRE, ARTHUR, 25, South street, Park lane.

1864 NUNN, THOMAS WILLIAM, Surgeon to the Middlesex Hospital; 8, Stratford place, Oxford street.

Elected

1859 *Nunneley, Thomas, Senior Surgeon to the Leeds Eye and Ear Infirmary; Leeds. *Trans.* 3.

1847 O'Connor, Thomas, March, Cambridgeshire.

1843 O'Connor, William, M.D., Physician to the Royal Free Hospital; 30, Upper Montagu street, Montagu square.

1858 Ogle, John William, M.D., Assistant-Physician to, and Lecturer on Pathology at, St. George's Hospital; 13, Upper Brook street, Grosvenor square. *Trans.* 4.

1855 *Ogle, William, M.A., M.D., Physician to the Derby Infirmary; 3, Stewart terrace, Derby.

1860 Ogle, William, M.D., Lecturer on Physiology at St. George's Hospital, and Physician to the St. George's and St. James's Dispensary; 34, Clarges street, Piccadilly.

1850 Oldham, Henry, M.D., Obstetric Physician to, and Clinical Lecturer on Midwifery at, Guy's Hospital; 26, Finsbury square. C. 1865. *Trans.* 1.

1846 *Ormerod, Edward Latham, M.D., Physician to the Sussex County Hospital; 14, Old Steine, Brighton. *Trans.* 2.

1847 *Page, William Bousfield, Surgeon to the Cumberland Infirmary; Carlisle. *Trans.* 2.

1840 Paget, James, F.R.S., Surgeon Extraordinary to H.M. the Queen; Surgeon in Ordinary to H.R.H. the Prince of Wales; Surgeon to, and Lecturer on Surgery at, St. Bartholomew's Hospital, and Surgeon to Christ's Hospital; Member of the Senate of the University of London; 1, Harewood place, Hanover square. C.1848-9. V.P. 1861. *Trans.* 8. *Sci. Com.*

1858 *Paley, William, M.D., Physician to the Ripon Dispensary; Ripon, Yorkshire.

1861 Palfrey, James, M.D., Assistant Obstetric Physician to the London Hospital; Physician to the Metropolitan Free Hospital, and Obstetric Physician to the Farringdon General Dispensary and Lying-in Charity; 25, Finsbury place.

1836 Parker, Langston, Hon. Surgeon to the Queen's Hospital, Birmingham; Colmore row, Birmingham.

Elected

1847 PARKER, NICHOLAS, M.D., Physician to the London Hospital; 22, Finsbury square.

1841 PARKIN, JOHN, M.D., Rome.

1851 PART, JAMES, M.D., 7, Camden road villas, Camden town.

1828 †PARTRIDGE, RICHARD, F.R.S., Professor of Anatomy to the Royal Academy of Arts, Surgeon to King's College Hospital, and Professor of Anatomy in King's College, London; 17, New street, Spring gardens. S. 1832-6. C. 1837-8. V.P. 1847-8. C. 1861-2. P. 1863-4.

1845 PEACOCK, THOMAS BEVILL, M.D., Physician to, and Lecturer on Medicine at, St. Thomas's Hospital; Physician to the City of London Hospital for Diseases of the Chest, Victoria park; 20, Finsbury circus. S. 1855-6. *Trans.* 2.

1864 PEARSON, DAVID RITCHIE, M.D., 23, Upper Phillimore place, Kensington.

1856 PEIRCE, RICHARD KING, 16, Norland place, Notting hill.

1830 PELECHIN, CHARLES P., M.D., St. Petersburgh.

1855 *PEMBERTON, OLIVER, Surgeon to the Birmingham General Hospital, and Lecturer on Surgical Pathology at Sydenham College; 18, Temple row, Birmingham. *Trans.* 1.

1844 PETTIGREW, WILLIAM VESALIUS, M.D., Surgeon to the Female Orphan Asylum, Lambeth; 7, Chester street, Grosvenor place.

1848 PHILLIPS, EDWARD, M.D., F.L.S., Physician to the Coventry and Warwickshire Hospital; Coventry, Warwickshire.

1852 PHILLIPS, RICHARD, 52, Leinster square, Westbourne grove.

1854 PHILLIPS, THOMAS BACON, M.D., Physician to the Brighton and Hove Dispensary; 36, Lansdowne place, Brighton.

1846 PHILP, FRANCIS RICHARD, M.D., Colby House, Kensington, and Sherborne House, Harrogate, Yorkshire.

1851 *PICKFORD, JAMES HOLLINS, M.D., M.R.I.A., 1, Cavendish place, Brighton.

1836 PIDDUCK, ISAAC, M.D., Physician to the Bloomsbury Dispensary; 22, Montague street, Russell square. *Pro.*2.

Elected

1841 PITMAN, HENRY ALFRED, M.D., *Treasurer*, Physician to, and Lecturer on Medicine at, St. George's Hospital; Consulting Physician to the Royal General Dispensary, St. Pancras; 94, Gloucester place, Portman square, L. 1851-3. C. 1861-2. T. 1863-5.

1850 POLAND, ALFRED, Surgeon to, and Lecturer on Surgery at, Guy's Hospital and to the Eye Infirmary attached to the Hospital; 32, Trinity square, Southwark. C. 1865.

1845 POLLOCK, GEORGE DAVID, Surgeon in Ordinary to H.R.H. the Prince of Wales; Surgeon to St. George's Hospital; 27, Grosvenor street. C. 1856-7. L. 1859-62. *Trans.* 2.

1865 POLLOCK, JAMES EDWARD, M.D., Physician to the Hospital for Consumption, Brompton; 52, Upper Brook street, Grosvenor square.

1843 POPE, CHARLES, M.D., Glastonbury, Somersetshire.

1846 POTTER, JEPHSON, M.D., F.L.S., Physician to the Liverpool General Hospital for Consumption and Diseases of the Chest; 109, Upper Parliament street, Liverpool.

1842 POWELL, JAMES, M.B.

1851 POWER, ROBERT FRANCIS, M.D., 71, Gloucester place, Portman square.

1857 PRIESTLEY, WILLIAM OVEREND, M.D., Physician-Accoucheur to H.R.H. the Princess Louis of Hesse; Professor of Midwifery in King's College, London; and Physician for the Diseases of Women and Children to King's College Hospital; Examiner in Midwifery at the University of London; Consulting Physician-Accoucheur to the St. Marylebone Infirmary; 17, Hertford street, May fair. *Sci. Com.*

1839 PROPERT, JOHN, Consulting Surgeon to the Society of Ancient Britons; 6, New Cavendish street, Portland place.

1845 PYLE, JOHN, 59, Oxford terrace, Hyde park. C. 1860.

1850 QUAIN, RICHARD, M.D., Physician to the Hospital for Consumption and Diseases of the Chest; Member of the Senate of the University of London; 56, Harley street, Cavendish square. *Trans.* 1. *Sci. Com.*

Elected

1835 †QUAIN, RICHARD, F.R.S., Surgeon Extraordinary to H.M. the Queen; Professor of Clinical Surgery in University College, London, and Surgeon to University College Hospital; Consulting Surgeon to the Eye Infirmary attached to the Hospital; 32, Cavendish square. C. 1838-9. L. 1846-8. T. 1851-3. V.P. 1856-7. *Trans.* 1. *Pro.* 2.

1852 RADCLIFFE, CHARLES BLAND, M.D., Physician to, and Lecturer on Materia Medica at, the Westminster Hospital; Physician to the National Hospital for the Paralysed and Epileptic; 25, Cavendish square.

1857 RANKE, HENRY, M.D., Munich.

1854 RANSOM, WILLIAM HENRY, M.D., Physician to the Nottingham General Hospital; the Pavement, Nottingham.

1859 RAYMOND, HENRY HUNTER, Cirencester, Gloucestershire.

1858 REED, FREDERICK GEORGE, M.D., 46, Hertford street, May fair. *Trans.* 1.

1821 REEDER, HENRY, M.D.

1857 REES, GEORGE OWEN, M.D., F.R.S., Physician to, and Lecturer on Medicine at, Guy's Hospital; 26, Albermarle street, Piccadilly. *Trans.* 1.

1855 REYNOLDS, JOHN RUSSELL, M.D., Professor of Clinical Medicine at University College, London, and Physician to University College Hospital; Physician to the National Hospital for the Paralysed and Epileptic; 38, Grosvenor street.

1865 RHODES, GEORGE WINTER, Surgeon to the Huddersfield Infirmary; 30, Ramsden street, Huddersfield.

1847 RICHARDS, SAMUEL, M.D., 36, Bedford square.

1852 RICHARDSON, CHRISTOPHER THOMAS, M.B.

1849 *RICHARDSON, WILLIAM, M.D., 9, Ephraim road, Tunbridge Wells, Kent.

1845 RIDGE, BENJAMIN, M.D., 21, Bruton street, Berkeley square.

1843 RIDGE, JOSEPH, M.D., 39, Dorset square. C. 1858. *Pro.* 1.

1852 RIDLEY, CHARLES, Surgeon to the Royal Society for Protection of Life from Fire; 6, Charlotte street, Bedford square.

Elected

1863 RINGER, SYDNEY, M.D., Professor of Materia Medica at University College, and Assistant-Physician to University College Hospital; Assistant-Physician to the Hospital for Sick Children; 15, Cavendish place.

1864 RITCHIE, CHARLES GEORGE, M.D., 36, Mount street, Grosvenor square.

1852 ROBERTS, JOHN, M.R.C.P., 75, Grosvenor street.

1855 ROBERTSON, CHARLES ALEXANDER LOCKHART, M.D., Medical Superintendent of the Sussex County Lunatic Asylum; Hayward's Heath, Sussex.

1857 ROBERTSON, JOHN CHARLES GEORGE, Assistant Medical Officer, Female Department, Middlesex County Lunatic Asylum, Hanwell.

1862 ROBINSON, CHARLES, 11, Montagu st., Montagu sq.

1843 ROBINSON, GEORGE, M.D. *Trans.* 2.

1843 RODEN, WILLIAM, M.D., the Grange, Kidderminster, Worcestershire.

1835 ROE, GEORGE HAMILTON, M.D., Senior Physician to the Hospital for Consumption and Diseases of the Chest; 57, Park street, Grosvenor square. C. 1841-2. *Trans.* 1.

1836 ROGERS, ARNOLD, Consulting Surgeon-Dentist to St. Bartholomew's Hospital; 16, Hanover square.

1829 ROOTS, WILLIAM SUDLOW, F.L.S., Surgeon to the Royal Establishment at Hampton Court; Kingston, Surrey.

1850 ROPER, GEORGE, 180, Shoreditch.

1855 ROSCOW, THOMAS TATTERSALL, M.D.

1836 *ROSE, CALEB BURRELL, F.G.S., 25, King street, Great Yarmouth, Norfolk. *Trans.* 1.

1857 ROSE, HENRY COOPER, M.D., High street, Hampstead.

1849 ROUTH, CHARLES HENRY FELIX, M.D., Physician to the Samaritan Free Hospital for Women and Children; 52, Montagu square. *Trans.* 1.

1863 ROWE, THOMAS SMITH, M.D., Surgeon to the Royal Sea-Bathing Infirmary; Cecil street, Margate, Kent.

1834 RUMSEY, HENRY WYLDBORE, Wolseley house, Cheltenham.

1845 RUSSELL, JAMES, M.D., Physician to the Birmingham General Hospital, and Lecturer on Pathology and Therapeutics at Sydenham College; 91, New Hall street, Birmingham.

Elected

1851 SALTER, HENRY HYDE, M.D., F.R.S., Assistant-Physician to, and Lecturer on Physiology and Pathology at, the Charing Cross Hospital; 6, Montague street, Russell square. *Sci. Com.*

1856 SALTER, SAMUEL JAMES A., F.R.S., F.L.S., Surgeon-Dentist to, and Lecturer on Dental Surgery at, Guy's Hospital; 17, New Broad street, City. *Trans.* 2.

1849 SANDERSON, HUGH JAMES, M.D., Physician to the Hospital for Women; 26, Upper Berkeley street, Portman square.

1855 SANDERSON, JOHN BURDON, M.D., Assistant-Physician to the Middlesex Hospital; Medical Officer of Health for Paddington; 49, Queen Anne street, Cavendish square. *Trans.* 1. *Sci. Com.*

1847 SANKEY, WILLIAM HENRY OCTAVIUS, M.D., Lecturer on Mental Diseases at University College, London; Sandywell park, near Cheltenham.

1845 SAUNDERS, EDWIN, Surgeon-Dentist to H.M. the Queen, and to H.R.H. the Prince of Wales; 13A, George street, Hanover square.

1834 SAUVAN, LUDWIG V., M.D., Warsaw.

1859 SAVORY, WILLIAM SCOVELL, F.R.S., Assistant-Surgeon to, and Lecturer on General Anatomy and Physiology at, St. Bartholomew's Hospital; Examiner in Physiology and Comparative Anatomy at the University of London; 23A, Brook street, Grosvenor sq. *Trans.* 3. *Sci. Com.*

1853 SCHULHOF, MAURICE, M.D., Physician to the Royal General Dispensary, Bartholomew Close; 14, Brook street, Grosvenor square.

1861 *SCOTT, WILLIAM, M.D., Physician to the Huddersfield Infirmary; 12, New North road, Huddersfield.

1858 *SCRATCHLEY, GEORGE, M.D., New Orleans, Louisiana, U.S.

1863 SEDGWICK, WILLIAM, Surgeon to the St. Marylebone Provident Dispensary; 12, Park place, Upper Baker street.

1856 SERCOMBE, EDWIN, Surgeon-Dentist to St. Mary's Hospital; 49, Brook street, Grosvenor square. *Trans.* 1. *Pro.* 1.

1824 †SEYMOUR, EDWARD JAMES, M.D., F.R.S., Consulting Physician to the Seamen's Hospital Ship 'Dreadnought'; 13, Charles street, Berkeley square. C. 1826, 1831. S. 1827-8. V.P. 1830, 1842. *Trans.* 2.

Elected

1840 SHARP, WILLIAM, M.D., F.R.S., Horton House, Rugby. *Trans.* 1.

1837 †SHARPEY, WILLIAM, M.D., F.R.S., LL.D., Professor of Anatomy and Physiology in University College, London; Member of the Senate of the University of London, and Secretary of the Royal Society; 33, Woburn place, Russell square. C. 1848-9. V.P. 1862.

1836 †SHAW, ALEXANDER, Surgeon to, and Lecturer on Surgery at, the Middlesex Hospital; 25, Henrietta street, Cavendish square. C. 1842. S. 1843-4. V.P. 1851-2. T. 1858-60. *Trans.* 4.

1848 *SHEARMAN, EDWARD JAMES, M.D., Rotherham, Yorkshire.

1859 SIBLEY, SEPTIMUS WILLIAM, Lecturer on Pathological Anatomy at the Middlesex Hospital; 12, New Burlington street. *Trans.* 3. *Sci. Com.*

1849 SIBSON, FRANCIS, M.D., F.R.S., Physician to St. Mary's Hospital; Member of the Senate of the University of London; 40, Brook street, Grosvenor square. C. 1863-4. *Trans.* 1. *Sci. Com.*

1848 SIEVEKING, EDWARD HENRY, M.D., Physician in Ordinary to H.R.H. the Prince of Wales; Physician to, and Lecturer on Materia Medica at, St. Mary's Hospital; Physician to the National Hospital for the Paralysed and Epileptic; 17, Manchester square. C. 1859-60. S. 1861-3. *Trans.* 2. *Sci. Com.*

1842 SIMON, JOHN, F.R.S., *Vice-President*, Surgeon to, and Lecturer on General Pathology at, St. Thomas's Hospital; Medical Officer of the Privy Council; 8, Richmond terrace, Whitehall. C. 1854-55. V.P. 1865. *Trans* 1.

1865 SIMS, J. MARION, M.D., 1, Bolton row, May fair.

1857 SIORDET, JAMES LEWIS, M.B., Mentone.

1827 SKENE, GEORGE ROBERT, Bedford.

1824 †SKEY, FREDERIC CARPENTER, F.R.S., Consulting Surgeon to, and Lecturer on Clinical Surgery at, St. Bartholomew's Hospital; 24, Mount street, Grosvenor square. C. 1828. L. 1829-31. V.P. 1841-2. P. 1859-60. *Trans.* 1.

Elected

1852 SMITH, CHARLES CASE, Consulting Surgeon to the Suffolk General Hospital; Bury St. Edmund's, Suffolk.

1835 SMITH, JOHN GREGORY, Harewood, Leeds, Yorkshire.

1865 SMITH, PETER, M.D., 29, Wimpole street, Cavendish square.

1843 SMITH, ROBERT WILLIAM, M.D., M.R.I.A., Professor of Surgery in the University of Dublin; Surgeon to the Richmond Hospital; 63, Eccles street, Dublin.

1838 SMITH, SPENCER, *Treasurer*, Surgeon to, and Lecturer on Surgery at, St. Mary's Hospital; 9, Queen Anne street, Cavendish square. C. 1854. S. 1855-8. V.P. 1859-60. T. 1865.

1863 SMITH, THOMAS, Assistant-Surgeon to, and Demonstrator of Anatomy at, St. Bartholomew's Hospital, and Assistant-Surgeon to the Hospital for Sick Children; 7, Montague street, Russell square. *Trans.* 1.

1864 *SMITH, THOMAS HECKSTALL, Rowlands, St. Mary Cray, Kent.

1845 SMITH, WILLIAM, Surgeon to the Chesterfield and North Derbyshire Hospital and Dispensary, Chesterfield, Derbyshire. *Trans.* 1.

1847 SMITH, WILLIAM, M.D., Consulting Physician to the Weymouth Infirmary; Weymouth, Dorsetshire.

1850 SMITH, WILLIAM TYLER, M.D., Physician-Accoucheur to, and Lecturer on Midwifery at, St. Mary's Hospital; 21, Upper Grosvenor street. *Trans.* 2.

1851 SODEN, JOHN, Surgeon to the Bath United Hospital, and Consulting Surgeon to the Bath Eye Infirmary; 24, Circus, Bath. *Trans.* 2.

1830 †SOLLY, SAMUEL, F.R.S., Surgeon to St. Thomas's Hospital; Consulting Surgeon to the Royal General Dispensary, Bartholomew Close; 6, Savile row. L. 1838-40. C. 1845-6. V.P. 1849-50. *Trans.* 6.

1865 SOUTHAM, GEORGE, Surgeon to the Manchester Royal Infirmary; 21, Lever street, and Oakfield, Pendleton, Manchester. *Trans.* 1.

1865 SOUTHEY, REGINALD, M.B., Assistant-Physician to St. Bartholomew's Hospital; Physician to the Royal General Dispensary; 32, Montague place, Russell square.

Elected

1844 SPACKMAN, FREDERICK R., M.D., Harpenden, St. Albans.

1834 SPARK, JAMES, Italy.

1851 SPITTA, ROBERT JOHN, M.B., Medical Officer to the Clapham General Dispensary; Clapham, Surrey. *Trans.* 1.

1843 *SPRANGER, STEPHEN, Hursley, Hampshire.

1857 STANTON, JOHN, M.D., 9, Montagu square.

1851 STARTIN, JAMES, Surgeon to, and Lecturer on Cutaneous Disorders at, the Hospital for Diseases of the Skin, Blackfriars; 3, Savile row, Burlington gardens.

1854 STEVENS, HENRY, M.D. Lond., 78, Grosvenor street.

1842 STEWART, ALEXANDER PATRICK, M.D., *Librarian*, Physician to, and Lecturer on Medicine at, the Middlesex Hospital; 74, Grosvenor street. C. 1856-7. L. 1863-5.

1859 STEWART, WILLIAM EDWARD, 12, Weymouth street, Portland place.

1856 STOCKER, ALONZO HENRY, M.D., Resident Medical Superintendent of Grove Hall Lunatic Asylum, Bow.

1865 STOKES, WILLIAM, Jun., M.D., 5, Merrion square north, Dublin.

1843 STORKS, ROBERT REEVE, Paris.

1858 †STREATFEILD, JOHN FREMLYN, Assistant-Surgeon to the Royal London Ophthalmic Hospital, Moorfields, and Assistant-Surgeon to the Eye Infirmary attached to University College Hospital; 15, Upper Brook street, Grosvenor square.

1863 STURGES, OCTAVIUS, M.B., Physician to the Chelsea, Brompton, and Belgrave Dispensary; 85, Wimpole street.

1839 SUTHERLAND, ALEXANDER JOHN, M.D., F.R.S., Consulting Physician to the Royal South London Dispensary; 6, Richmond terrace, Whitehall. C. 1850-1. V.P. 1858-9. *Trans.* 1.

1860 SUTRO, SIGISMUND, M.D., Senior Physician to the German Hospital; 37A, Finsbury square.

1855 SUTTON, JOHN MAULE, M.D., Bloomfield, Narberth, Pembrokeshire.

1861 *SWEETING, GEORGE BACON, King's Lynn, Norfolk.

Elected

1842 SYME, JAMES, F.R.S.E., Surgeon in Ordinary to H.M. the Queen in Scotland; Professor of Clinical Surgery in the University of Edinburgh, and Surgeon to the Edinburgh Royal Infirmary; 2, Rutland street, Edinburgh. *Trans.* 5.

1854 *SYMONDS, FREDERICK, Surgeon to the Radcliffe Infirmary, and Consulting Surgeon to the Oxford Dispensary; 32, Beaumont street, Oxford.

1844 TAMPLIN, RICHARD WILLIAM, Surgeon to the Royal Orthopædic Hospital; 33, Old Burlington street.

1848 TANNER, THOMAS HAWKES, M.D., F.L.S., 9, Henrietta street, Cavendish square.

1864 TAUSSIG, GABRIEL, M.D., 70, Piazza Barberini, Rome.

1852 TAYLOR, ROBERT, Surgeon to the Central London Ophthalmic Hospital, and to the Cripple's Home, Hill street; 21, Edwards street, Portman square.

1845 TAYLOR, THOMAS, Lecturer on Chemistry at the Middlesex Hospital Medical School; 4, Vere st., Cavendish square.

1856 TEALE, THOMAS PRIDGIN, F.R.S., F.L.S., Surgeon to the Leeds General Infirmary; Albion st., Leeds. *Trans.* 2.

1859 TEGART, EDWARD, Junior, 49, Jermyn street, St. James's.

1862 THOMPSON, EDMUND SYMES, M.D., Assistant-Physician to King's College Hospital, and to the Hospital for Consumption, Brompton; 3, Upper George street, Portman square.

1857 THOMPSON, HENRY, M.D., Physician to, and Lecturer on Materia Medica at, the Middlesex Hospital; 52, Welbeck street, Cavendish square.

1852 THOMPSON, HENRY, Surgeon Extraordinary to H.M. the King of the Belgians, Surgeon to University College Hospital, and Consulting Surgeon to the St. Marylebone Infirmary; 35, Wimpole street, Cavendish square. *Trans.* 3.

1862 THOMPSON, REGINALD EDWARD, M.D., Physician to the St. George's and St. James's Dispensary; 21, South street, Park lane.

1836 THURNAM, JOHN, M.D., Resident Medical Superintendent of the Wilts County Asylum, Devizes, Wiltshire. *Trans.* 4.

Elected

1848 TILT, EDWARD JOHN, M.D., Consulting Physician to the Farringdon General Dispensary and Lying-in Charity; 60, Grosvenor street.

1828 TORRIE, JAMES, M.D., Old Bridge of Don, by Aberdeen.

1843 TOYNBEE, JOSEPH, F.R.S., Aural Surgeon to, and Lecturer on Aural Surgery at, St. Mary's Hospital; Consulting Aural Surgeon to the Asylum for the Deaf and Dumb, and to the St. George's and St. James's Dispensary; 18, Savile row, Burlington gardens. C. 1863-4. *Trans.* 9. *Pro.* 1.

1850 TRACY, SAMUEL JOHN, Surgeon-Dentist to St. Bartholomew's and Christ's Hospitals; 28, Old Burlington street.

1859 TRUMAN, EDWIN THOMAS, Surgeon-Dentist in Ordinary to Her Majesty's Household; 23, Old Burlington street.

1864 TUFNELL, THOMAS JOLLIFFE, Examiner in Surgery to the Royal College of Surgeons of Ireland; 58, Lower Mount street, Merrion square, Dublin.

1862 TUKE, THOMAS HARRINGTON, M.D., Manor House, Chiswick, and 37, Albemarle street.

1855 TULLOCH, JAMES STEWART, M.D., 1, Pembridge place, Bayswater.

1864 TURNER, GEORGE, 37, Sussex Gardens, Hyde park, W.

1845 TURNER, THOMAS, F.L.S., Consulting Surgeon to the Manchester Royal Infirmary, and Lecturer on Anatomy and Physiology at the Manchester Royal School of Medicine; 77, Mosley street, Manchester.

1846 URE, ALEXANDER, Surgeon to, and Lecturer on Clinical Surgery at, St. Mary's Hospital, and Consulting Surgeon to the Westminster General Dispensary; 18, Upper Seymour street, Portman square. *Trans.* 1.

1806 VAUX, BOWYER, Teignmouth, Devon.

1865 VERNON, BOWATER JOHN, Assistant Demonstrator, St. Bartholomew's Hospital.

1828 VULPES, BENEDETTO, M.D., Physician to the Hospital of Aversa, and the Hospital of Incurables, Naples.

1854 WADDINGTON, EDWARD, Kettlethorpe Hall, Newark, Notts.

1841 WADE, ROBERT, Senior Surgeon to the Westminster General Dispensary; 68, Dean street, Soho. *Trans.* 1.

Elected

1864 WAITE, CHARLES DERBY, M.B., 3, Old Burlington street.

1861 *WALSH, JAMES, M.D., Staff-Surgeon, R.N., 41, Cathrine street, Limerick, Ireland.

1852 WALSHE, WALTER HAYLE, M.D., Emeritus Professor of the Principles and Practice of Medicine, University College, London; Consulting Physician to the Hospital for Consumption; 37, Queen Anne st., Cavendish sq. *Trans.* 1.

1851 WALTON, HENRY HAYNES, Surgeon to the Central London Ophthalmic Hospital, and Surgeon to St. Mary's Hospital; 69, Brook street, Hanover sq. *Trans.* 1. *Pro.* 1.

1852 WANE, DANIEL, M.D., 20, Grafton street, Berkeley square.

1821 WARD, WILLIAM TILLEARD.

1858 WARDELL, JOHN RICHARD, M.D., 4, Belmont, Tunbridge Wells.

1846 WARE, JAMES THOMAS, Consulting Surgeon to the Finsbury Dispensary, and Hon. Surgeon to the Metropolitan Convalescent Institution; 18, Gordon square.

1818 WARE, JOHN, Clifton Down, near Bristol.

1814 †WARE, MARTIN, 18, Gordon square. C. 1844-5. T. 1846. V.P. 1853.

1829 WARRY, ELIAS TAYLOR, M.D., Yeovil, Somerset.

1861 WATERS, A. T. HOUGHTON, M.D., Physician to the Liverpool Northern Hospital, and Lecturer on Anatomy and Physiology in the Liverpool Royal Infirmary School of Medicine; 27, Hope street, Liverpool. *Trans.* 3.

1837 WATSON, THOMAS, M.D., F.R.S, D.C.L., President of the Royal College of Physicians; Physician Extraordinary to H.M. the Queen; Consulting Physician to King's College Hospital; 16, Henrietta street, Cavendish sq. C. 1840-1, 1852. V.P. 1845-6.

1861 WATSON, WILLIAM SPENCER, M.B., Assistant-Surgeon to King's College Hospital; 27, Montague st., Russell sq.

1854 WEBB, WILLIAM, M.D., Wirksworth, Derbyshire.

1840 WEBB, WILLIAM WOODHAM, M.D., Cliff House, Kirtley, South Lowestoft, Suffolk.

1842 WEBER, FREDERIC, M.D., *Vice President*, 44, Green street, Park lane. C. 1857. V.P. 1865.

1857 WEBER, HERMANN, M.D., Physician to the German Hospital; 49, Finsbury square. *Trans.* 4.

Elected

1835 †WEBSTER, JOHN, M.D., F.R.S., Physician to the Scottish Hospital, and Consulting Physician to the St. George's and St. James's Dispensary; 20, Brook street, Grosvenor square. C. 1843-4. V.P. 1855-6. *Trans.* 6. *Pro.* 1.

1844 WEGG, WILLIAM, M.D., Physician to the St. George's and St. James's Dispensary; 26, Park lane. L. 1854-8. C. 1861-2.

1861 WELLS, JOHN SOELBERG, Professor of Ophthalmology in King's College, London, and Ophthalmic Surgeon to King's College Hospital; 16, Savile row.

1854 WELLS, THOMAS SPENCER, Surgeon in Ordinary to H.M.'s Household; Surgeon to the Samaritan Free Hospital for Women and Children; 3, Upper Grosvenor street. *Trans.* 4. *Pro.* 1.

1842 †WEST, CHARLES, M.D., Physician to the Hospital for Sick Children; 61, Wimpole street, Cavendish square. C. 1855-6. V.P. 1863. *Trans.* 2. *Sci. Com.*

1841 WEST, THOMAS, M.D., Daventry, Northamptonshire.

1828 WHATLEY, JOHN, M.D.

1849 WHITE, JOHN.

1852 WIBLIN, JOHN, M.D., Medical Inspector of Emigrants and Recruits; Southampton. *Trans.* 1.

1844 WILDBORE, FREDERIC, 1, Trafalgar place east, [245] Hackney road.

1837 WILKS, GEORGE AUGUSTUS FREDERICK, M.D., 5, Lincoln's Inn Fields.

1863 WILKS, SAMUEL, M.D., Assistant-Physician to, and Lecturer on Medicine at, Guy's Hospital, Examiner in Medicine at the University of London; 11, St. Thomas's street, Southwark.

1865 WILLETT, ALFRED, Assistant-Surgeon to, and Warden of the College, St. Bartholomew's Hospital; the College, St. Bartholomew's Hospital.

1864 WILLETT, EDMUND SPARSHALL, M.D., Resident Physician, Wyke House, Isleworth, Middlesex.

1860 WILLIAMS, ARTHUR WYNN, M.D., Physician-Accoucheur, Western General Dispensary; 20, King st., Portman sq.

Elected

1840 WILLIAMS, CHARLES JAMES BLASIUS, M.D., F.R.S., Consulting Physician to the Hospital for Consumption; 49, Upper Brook street, Grosvenor square. C. 1849-50. V.P. 1860-1. *Sci. Com.*

1859 *WILLIAMS, CHARLES, Surgeon to the Norwich Dispensary 2, Bank Plain, Norwich.

1859 WILLIAMS, JOSEPH, M.D., 8, Tavistock square.

1829 WILLIS, ROBERT, M.D., Barnes, Surrey. L. 1838-41.

1839 †WILSON, ERASMUS, F.R.S., Consulting Surgeon to the St. Pancras Infirmary; 17, Henrietta street, Cavendish square. *Trans.* 2.

1863 WILSON, ROBERT JAMES, L.R.C.P. Edinb., 24, Grand Parade, St. Leonards-on-Sea, Sussex.

1850 *WISE, ROBERT STANTON, M.D., Consulting Physician to the Southam Eye and Ear Infirmary; Banbury, Oxford-sh.

1825 WISE, THOMAS ALEXANDER, M.D., Rostellan Castle, Rostellan, County Cork.

1841 WOOD, GEORGE LEIGHTON, Surgeon to the Bath General Hospital; 27, Queen square, Bath.

1851 WOOD, JOHN, Assistant-Surgeon to King's College Hospital, and Demonstrator of Anatomy in King's College, London; 4, Montague street, Russell square. *Trans.* 2.

1848 WOOD, WILLIAM, M.D., Physician to St. Luke's Hospital for Lunatics; 54, Upper Harley street.

1843 WOODFALL, JOHN WARD, M.D., Physician to the West Kent Infirmary; Maidstone, Kent.

1833 †WORMALD, THOMAS, Surgeon to St. Bartholomew's Hospital; 42, Bedford Row. C. 1839. V.P. 1854.

1842 WORTHINGTON, WILLIAM COLLINS, Senior Surgeon to the Lowestoft Infirmary; Lowestoft, Suffolk. *Trans.* 3.

1848 WRIGHT, EDWARD JOHN, 13, Montague place, Clapham road.

1855 WRIGHT, HENRY G., M.D., Physician to the Samaritan Free Hospital for Women and Children; 23, Somerset street, Portman square.

1860 WYATT, JOHN, Surgeon-Major, Coldstream Guards; Hospital, Vincent square, Westminster.

[It is particularly requested, that any change of Title, Appointment, or Residence, may be communicated to the Secretaries before the 1st of October in each year, in order that the List may be made as correct as possible.]

HONORARY FELLOWS.

(Limited to Twelve.)

Elected

1841 BRANDE, WILLIAM THOMAS, D.C.L., F.R.S., Hon. Professor of Chemistry at the Royal Institution of Great Britain, Member of the Senate of the University of London; Royal Mint, Tower hill, and 15, Calverley park, Tunbridge Wells.

1835 BREWSTER, SIR DAVID, K.H., D.C.L., LL.D., F.R.S., Corresp. Memb. Institute of France, Principal and Vice-Chancellor of the University of Edinburgh.

1853 BRODIE, SIR BENJAMIN COLLINS, Bart., M.A., F.R.S., Aldrichian Professor of Chemistry in the University of Oxford; Cowley House, Oxford.

1847 CHADWICK, EDWIN, late Commissioner of the Board of Health.

1862 DAUBENY, CHARLES GILES BRIDLE, LL.D., M.D., F.R.S., Hon. M.R.I.A., &c., Professor of Botany, Oxford.

1835 FARADAY, MICHAEL, D.C.L., F.R.S., Corresp. Memb. Institute of France, Member of the Senate of the University of London, and Fullerian Professor of Chemistry in the Royal Institution.

1857 FARR, WILLIAM, M.D., D.C.L., F.R.S., General Register Office, Somerset House, and Southlands, Bromley, Kent.

1841 HERSCHEL, SIR JOHN FREDERICK WILLIAM, Bart., D.C.L., F.R.S., Corresp. Memb. Institute of France; Collingwood, near Hawkhurst, Kent.

1847 OWEN, RICHARD, D.C.L., LL.D., F.R.S., Corresp. Memb. Institute of France (Foreign Associate of the Academy of Sciences); Superintendent of the Natural History Departments in the British Museum; Sheen Lodge, Mortlake.

1825 SEDGWICK, The Rev. ADAM, A.M., D.C.L., F.R.S., Woodwardian Professor of Geology, Cambridge.

1841 WHEWELL, The Rev. WILLIAM, D.D., F.R.S., Master of Trinity College, Cambridge.

FOREIGN HONORARY FELLOWS.

(Limited to Twenty.)

Elected

1841 ANDRAL, G., M.D., Member of the Institute and of the Imperial Academy of Medicine, Physician in Ordinary to the Emperor of the French, Professor of Pathology in the Faculty of Medicine ; Paris.

1862 CRUVEILHIER, JEAN, M.D., Physician to the " Hôpital de la Charité," Professor of Pathological Anatomy to the Faculty of Medicine, Member of the Imperial Academy of Medicine, &c. ; Paris.

1864 DONDERS, F.C., M.D., Professor of Physiology and Ophthalmology at the University of Utrecht.

1856 DUBOIS, BARON PAUL, Commander of the Legion of Honour, Member of the Imperial Academy of Medicine, late Dean of the Faculty of Medicine; Paris.

1835 EKSTRÖMER, CARL JOHAN, M.D., C.M., K.P.S., and W., Physician to the King of Sweden, President of the College of Health, and Director General of Hospitals; Stockholm.

1841 EHRENBERG, CHRISTIAN GOTTFRIED, Member of the Institute of France; Berlin.

1859 HENLE, J. M.D., Professor of Anatomy at Göttingen.

1841 JACKSON, JAMES, M.D., LL.D., Emeritus Professor of Medicine in the Harvard University, Boston, U.S.

1856 LANGENBECK, BERNHARD, M.D., Professor of Surgery in the University of Berlin.

1843 LIEBIG, BARON JUSTUS von, M.D., Foreign Associate of the Academy of Sciences, Conservator of the Royal Collection, and Professor of Chemistry in the University of Munich.

1841 LOUIS, P. C. A., M.D., Honorary Physician to the Hôtel-Dieu, Member of the Imperial Academy of Medicine ; Paris.

1847 MATTEUCCI, CARLO, Professor in the University of Pisa, Member of the Institute of France; Minister of Public Instruction in Italy.

Elected

1841 PANIZZA, BARTOLOMEO, M.D.; Pavia.

1862 PIROGOFF, NIKOLAUS, M.D., Professor of Surgery to the Medico-Chirurgical Academy in St. Petersburg, Director of the Anatomical Institute, Consulting Physician to the Hospitals Obuchow, Peter-Paul, and Maria Magdalena; St. Petersburg.

1859 RAYER, PIERRE, M.D., Grand Officer of the Legion of Honour, Member of the Institute, and of the Imperial Academy of Medicine; Paris.

1850 ROKITANSKY, CARL, M.D., Curator of the Imperial Pathological Museum, and Professor of the University of Vienna. Referee for Medical and University Education to the Austrian Ministry.

1856 STROMEYER, LOUIS, M.D., Director-General of the Medical Department of the Army of Hanover; Hanover.

1856 VELPEAU, A., Member of the Institute, and of the Imperial Academy of Medicine, Professor in the Faculty of Medicine, Surgeon to the "Hôpital de la Charité," President of the Academy of Sciences; Paris.

1856 VIRCHOW, RUDOLPH, M.D., Professor of Pathological Anatomy in the University of Berlin.

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REMARKABLE INSTANCE

OF A

GROWTH SPRINGING FROM THE EPIGLOTTIS,

WHICH WAS SUCCESSFULLY REMOVED WITH THE AID OF THE LARYNGOSCOPE.

BY

GEORGE DUNCAN GIBB, M.D., LL.D., M.R.C.P.,

ASSISTANT-PHYSICIAN TO, AND LECTURER ON FORENSIC MEDICINE AT, THE WESTMINSTER HOSPITAL.

Received October 13th.—Read November 8th, 1864.

Although the pathology of the epiglottis has been greatly enriched since the revival of the laryngoscope, tumours or growths springing directly from the cartilage itself are very uncommon. This is proved by an examination of the rich museums of the metropolis, wherein the rarity of epiglottic growths becomes quite apparent. After an extensive experience of affections involving the throat and larynx, I must acknowledge, if diseases of the cartilage arising from inflammation or other causes be excluded, that offshoots or growths are seldom to be seen. However, on a careful scrutiny of all the museums in London, and in many of the provincial towns, I have met with a few instances of tumours growing in the vicinity of the epiglottis, and not unfrequently involving the cartilage, or so compressing it

downwards as ultimately to destroy life. And in some of the London museums examples may be seen of vegetations extending to the laryngeal surface of the cartilage; such cases are noticed in the "Table of Growths" in the second edition of my work on 'Diseases of the Throat,' lately published, and need not be recapitulated here. In one of these cases (No. 14) the posterior surface of the epiglottis of a child, preserved in University College Museum, is studded with warty vegetations; in another (No. 16), in St. Bartholomew's Museum, a flat spongy growth of a medullary nature springs from the laryngeal surface of the epiglottis and superior orifice of the larynx of an adult. The case of mucous cyst of the laryngeal surface of the epiglottis brought before this Society at its last session by that experienced observer, Mr. Durham, will be in the recollection of the Fellows. In all these instances the laryngeal and not the lingual surface of the cartilage was affected.

Any addition, therefore, to our information upon an important and dangerous form of disease, more particularly involving the lingual surface of the epiglottis, must necessarily prove acceptable. It is just possible that, now that we have the laryngoscope to aid us, other and perhaps similar examples to that now brought before this Society may present themselves. It is probable that such cases may have occurred in the practice of others, but they could hardly have escaped detection, as the impediments they would offer both to breathing and swallowing would have directed attention to the throat, and their discovery would have been rendered all but certain, even without an acquaintance with laryngoscopy. So far as the evidence at present extends, we are justified in concluding that epiglottic swellings, resulting from morbid growths or otherwise, are rare phenomena in medicine, and yet when they do arise an error in diagnosis is likely to lead us to look upon any measures resorted to for relief as almost hopeless, for the impression would be that the œsophagus or larynx is so involved that all interference would be not only useless, but positively hazardous.

With the view, therefore, of placing upon record a case of

great practical value and interest, and at the same time showing what may be done under circumstances apparently most urgent, the following case is brought before this Society. It shows very satisfactorily how some of the great functions of life—swallowing, for instance—may be affected by causes wholly distinct from those at first suspected.

The particulars of the case shall be first narrated, and a general commentary will then be offered upon it.

Mrs. S. B—, æt. 60, the wife of a clergyman, consulted me on July 5th, 1864. Her son, a medical gentleman, who lived at a distance from her, wrote to tell me that she had suffered from her throat for two years, nothing having been detected but chronic inflammation. He was convinced, he said, from her symptoms, that there was stricture of the œsophagus. For twelve months matters had been getting worse; in addition to the presence of dysphagia he had heard of great pain and bloody expectoration, and was afraid that the stricture was malignant and in a state of ulceration. This induced him to pay her a visit, when he discovered a polypoid tumour, the size of a small walnut, at the back of the mouth. He pointed this out to her medical attendant, and recommended its immediate removal. She proposed coming to London to consult me. How far the polyp (he wrote) has to do with œsophageal narrowing, will be interesting to know.

I attached much importance to this gentleman's opinion, for he had himself removed two pharyngeal tumours by a double canula and wire through the nose, and was about to remove another.

On presenting herself to me I found her pale and wan, and somewhat emaciated; she spoke in a low, thick, guttural tone, as if the mouth was full of food; indeed, the character of the voice was peculiar in this respect, and clearly pointed to influences wholly above the glottis; dysphagia was complete with fluids, but she could swallow a little farinaceous food, such as Indian corn flour, milk and arrow-root, and a little soft bread, or an egg. In attempting to swallow fluid it would not pass downwards, and frequently came through the nostrils. She had not had any dyspnœa, except when she

lay on her back in bed at night. She coughed and expectorated during the whole of the night, but not in the daytime. The expectoration was mucous and frothy, sometimes mixed with blood, at times thick and stringy.

Inspection with the laryngoscope.—A distinct roundish, prominent, and projecting tumour, the size of a small walnut, was seen occupying the position of a pendent epiglottis, which during the act of swallowing became elevated, and could be seen with the unaided eye at the back of the mouth, pressing against the pharynx. The epiglottis itself was not seen, nor the interior of the larynx, but the posterior margins of both arytenoid cartilages could be observed. It seemed as if the tumour—partly divided by a sulcus antero-posteriorly—grew from the lingual surface of the epiglottis. The anterior surface and right border of the tumour were red and vascular, in some parts of a whitish colour and eroded, giving rise to free expectoration of mucus and some oozing of blood. The breathing was unaffected.

Seeing the nature of the case, I obtained the assistance of Mr. Ure, and again examined the throat in his presence; he readily recognised all that has been described, with the utmost distinctness, and agreed with me in the propriety of removing the tumour. Indeed, there was nothing in the patient's history, family or otherwise, to contra-indicate it; the appearance of the tumour, too, although suspicious, was not malignant, partaking somewhat of a spongy character. Believing that the entire cartilage was involved in the disease, my intention was to remove the whole mass, including all the free portion of the epiglottis, and then to trust to the efforts of nature so to contract the upper laryngeal orifice as to permit of swallowing without inconvenience, as happens in cases where the free portion of the cartilage has been destroyed by ulceration or accident. The patient's spirits were perfectly good, and she was prepared to undergo anything for relief.

On the 6th my laryngoscopic inspection was even clearer than the day before, yet more of the true epiglottis was seen. The interior of the larynx was nearly observed twice.

Fig. 1 gives the view of the tumour with the unaided eye at the back of the mouth. (See woodcut.)

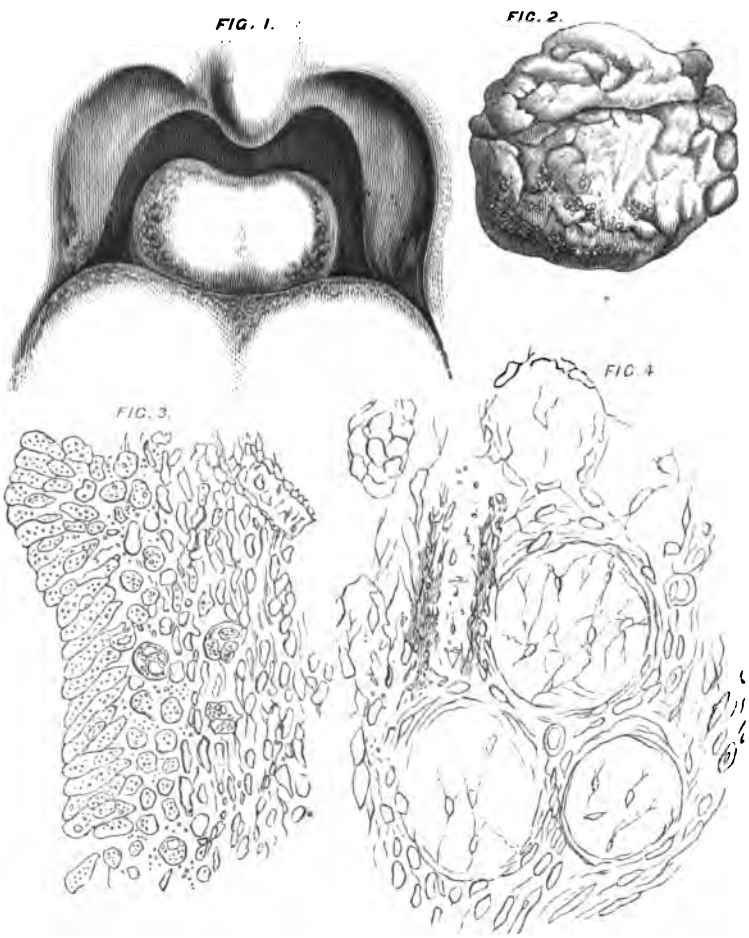

FIG. 1.—View of the tumour, with the unaided eye, at the back of the mouth.

FIG. 2.—Upper surface of the tumour, showing its actual size and shape.

FIGS. 3 and 4.—Microscopic appearances of the tumour from the centre and edge of the section.

7th.—At 3 o'clock p.m. I proceeded to remove the growth. The room was darkened and a moderator lamp placed upon a table towards the left of the patient, who was comfortably seated in a chair higher than the one used by myself; her head was steadied by Mr. Philps, who stood behind her; Dr. Logan was seated at her right side, and held her tongue out; whilst Mr. Ure, standing to my left, was ready with a pair of long vulsellum forceps to seize the tumour at the back of the mouth the moment it was detached. Mr. Holthouse and the lady's husband were also present.

A frontal mirror was used, and worn just above the nose, to permit of a central uninterrupted light and the employment of both eyes freely. Chloroform was not given, and there was not any undue sensibility about the throat and fauces. Everything being prepared, the laryngeal mirror was introduced and the growth was readily seen by every one present. I then steadied the light and introduced the mirror into the mouth with my left hand, and with my right the thick loop of wire and shank of the écraseur; the loop of wire was passed over the tumour, but an immediate effort at retching occurred, and the tumour at once slipped out. Seeing this, I dispensed with the little mirror, pressed down the tongue at its middle with my left forefinger, passed the loop of wire in a second time, snared the growth to its very base, and at once drew the wire home and detached the tumour; at the same instant of time Mr. Ure passed in the vulsellum forceps and as quickly removed it from the mouth.

A little coughing ensued, with expectoration of blood, which was lessened by gargling with cold-water. As it was a little persistent, the mirror was introduced and blood was seen to exude from the entire lingual surface of the epiglottis, from which the tumour had been removed as cleanly as if by a knife; the denuded surface possessed a scarlet redness, and its margins were well defined. I immediately brushed this with a strong solution of tannin, with great advantage, the mirror enabling me to guide the brush.

The voice, from being thick and guttural, was at once altered to a smoother and more agreeable tone, and swallow-

ing could be attempted in small quantities, and a glass of wine and water was got down in this way. The firmness and great presence of mind displayed by the lady herself greatly contributed to the success and celerity of the operation. A gargle and drink of decoction of barley were prescribed, and rest in the recumbent posture.

The tumour became a little flattened after removal; its diameter was that of a small walnut, but not so thick; it was softish and spongy; variegated in colour, being a mixture of opaque white, purple, and red, and apparently very vascular. It did not possess a malignant aspect, but seemed epithelial, springing apparently from the mucous membrane of the lingual surface of the epiglottis alone. Although by no means pedunculated, its base was not as large as the growth itself, probably a fourth less, which was the fortunate circumstance which admitted of the wire loop passing between it and the epiglottis. The upper and posterior surfaces, towards the right, were of a mouldy colour; it is shaded dark in fig. 2 (see woodcut, p. 5), which is a sketch of its actual size and shape, and shows its upper surface.

As previously mentioned, I had intended, and, indeed, actually expected, to have taken away the entire free portion of the epiglottis, but fortunately it turned out otherwise. I had provided myself, therefore, with various appliances to arrest hæmorrhage, or obviate any other inconvenience that might have arisen in the event of removal of the epiglottis. I was ably assisted by all present, and am much indebted to Mr. Ure for his prompt and speedy removal of the growth from the mouth after its detachment with the écraseur, else its lodgment might have proved detrimental to the patient.

8.30 p.m.—Found her going on well, but as the barley decoction proved a little thick, a gargle was prescribed of tannin ʒij, syrup of roses ʒij, and water ʒviij. This seemed the more advisable, as there was still some oozing of blood. I advised, also, a teacupful of bread and milk at bedtime, to induce sleep, as she had had no rest during the previous night.

8th.—Slept well, and to-day is quite comfortable; she can swallow much better, but necessarily with a feeling of some

soreness. She was eating more, too, and had taken a little soft bread. Was sitting up as usual, and not confined to bed.

11th.—She called upon me, and complained of a little soreness and pain in the left ear and temples, as before the operation; she eats better, but has not tried anything solid. Cough still rather troublesome. The laryngeal mirror showed the cicatrix narrowing on the surface of the epiglottis. The right side still seemed pretty thick. Applied a solution of tannin, and prescribed a gargle of bromide of ammonium, alum, glycerine, and water.

12th.—Freely applied a solution of the iodide of silver in ether and water, in the presence of Mr. E. J. Lambart, of Paris. Was enabled to see the interior of the larynx. General health improving, takes more food and eats better; is not so low nor so thin as she was, and feels less pain.

13th.—Has still some pain in the left ear, extending from the throat. Nothing is to be seen at the back of the mouth on depressing the tongue with a depressor. During cicatrization of the wound a small projection, about the size of a large bean, slipped round, apparently from the right edge of the epiglottis, on to the surface of the cartilage. This I removed with my small laryngeal écraseur, and it came away in the loop of the wire; some bleeding followed. Afterwards I passed a probang down the œsophagus and found no stricture.

A small projection, about the size of a walnut, exists in the neck, externally, over the right side of the larynx, below and to the right of the cricoid cartilage. This had been present since she was nine years of age; it was rather hard, and at one time was supposed to cause stricture of the œsophagus.

15th.—She looks and eats better, but requires her food to be cold; has some slight soreness in swallowing. The wound and surrounding parts possess a good natural pink colour, and retraction is occurring forwards. Could see in the laryngeal mirror both arytenoids acting well, and nearly saw the larynx. Applied a solution of the nitrate of uranium. Has still a little cough at night, which is wholly epiglottic.

As she was to leave for home on the 17th, I prescribed a soothing and astringent gargle, with an excess of alum, to be increased if necessary, at the discretion of her ordinary medical attendant.

The tumour was examined by Dr. Andrew Clark, to whom I am indebted for the following note:

"The growth is essentially a connective tissue, though it contains other elementary textures, and is intensely vascular. The chief part of the tumour consists of an amorphous or slightly fibrillated stroma, containing branching and anastomosing connective tissue, corpuscles, nuclear fibres, and free nuclei; at one or two points the corpuscles are few; the matrix very abundant, soft, and infiltrated with a mucous fluid. At several other points, where the stroma is abundant, immature cartilage-corpuscles are present. Associated with these, but in a disorderly manner, are bits of muscular fibre, disintegrated racemose glands, and patches of cylindrical epithelium. The growth is unquestionably benign. In its present condition it may be called a connective-tissue growth. But that condition is not necessarily final; in fact, it is embryonic, and the elements entering into its composition might have become developed into any other growth. Most probably, however, it would have been a fibro-cartilaginous one. The question of its return is to be answered according to the completeness of the removal of the growth. If a considerable portion has been left it will in all probability, not only return, but return under the form of some higher structural development; but if it has been nearly all removed then the chances of complete recovery are great."

Figs. 3 and 4 (see woodcut, p. 5) show the microscopic appearances of the tumour examined by Dr. Clark, from the centre and edge of the section.

Commentary.—The dysphagia had been present on and off for two years, gradually increasing in its progressive severity, until at last but very little food could be swallowed. This, associated with great pain and bloody expectoration, led to the impression that there might be malignant contraction of the œsophagus, an impression which was strongly favoured

by the difficulty, if not impossibility (as I learnt), of passing a bougie. This is intelligible when the nature of the obstruction is considered, for the presence of a body of such a magnitude as that described, by its mere size and the spasms which it would excite, would effectually close that part of the pharynx immediately contiguous to it, and thus effectually bar the entrance of any foreign body introduced for exploration, just as it would food.

Although the presence of the growth had become known, yet the belief had been entertained that a stricture might be present as well, an opinion partly favoured by the swelling in the external part of the neck. Taking all things into account, I believed that there was no stricture, chiefly from the fact that even comparatively slight lesions, such as ulcers and moderate infiltration or inflammation of the epiglottis, are apt to cause troublesome dysphagia. Yet here was a large amount of disease to give rise to it.

Being satisfied in my own mind that disease of the epiglottis was the chief exciting cause of the dysphagia, the next step was to estimate the amount and extent of that disease, and to consider whether it was remediable by art or otherwise. Ordinary inspection furnished a certain amount of information, which was much increased by inspection with the laryngoscope. It was demonstrated to satisfaction that the epiglottis, and it alone, was the seat of the disease, commencing, as it were, in the valve, from its point of attachment to the tongue. The larynx was proved to be intact above the glottis by the character of the speech (phonation), which, although guttural and thick, was not in any manner laryngeal, *i. e.* emanating from a constricted or impaired glottis or larynx. There was at no time positive dyspnœa, unless from dorsal decubitus at night, when the pendent valve would, almost from its mere weight, close up the aperture of the larynx, and thus give rise to cough and bloody expectoration. There was fortunately no evidence of malignity, either in its appearance or otherwise; and although there was some expectoration of blood and mucus, the friction to which the part was subjected, together with the vascular and spongy nature

of the growth itself, would necessarily give rise to both. All my efforts, by inducing cough, hemming, forcible expiration, pressure on the tongue, or otherwise, failed in permitting of a view of the interior of the larynx. The diagnosis, nevertheless, was pretty clear, and the course to be pursued was obvious. Here was a growth whose nature and position were tolerably made out: what was to be done with it? The plan of its removal by caustic, Paris or Vienna paste, or, indeed, by any other mode short of detachment with a loop of wire, could not be entertained. Time and the condition of the patient, who was already becoming much emaciated, were both against mere local treatment. Indeed, there could be no doubt as to the propriety of removing the tumour, and its removal was determined upon after carefully weighing the risk and responsibility attaching to the separation of the whole of the free portion of the valve as far as its connection with the tongue. This measure was contemplated with a little anxiety, for reasons that must be obvious; but my anxiety subsided when I remembered that the cartilage had been shot and cut away, with full power over swallowing remaining, and that deglutition was compatible with an absence of the free portion of the epiglottis, as experience had demonstrated to me in seven patients, five of whom are still living. As the general health was otherwise good, no time was lost in carrying this out, and an instrument was extemporaneously devised for the purpose of snaring the growth, and at once cutting it off, as I had already done on eleven previous occasions within the larynx. Weiss and Son supplied me with the instrument used, and which consisted of a long canula (slightly curved at one end) and handle, employed, indeed, for another purpose, but excellently suitable for the object intended. A very thick twisted wire was introduced through the canula, allowing of a loop at the terminal end, capable of enclosing a good-sized walnut. The only objection I had to it was the extreme length of the canula, and the necessity of using both hands when the epiglottis and its tumour were snared. At the proper time, however, it was found to answer, as already

detailed in the narrative of the case, and effected its object most satisfactorily, producing exclamations of surprise and delight from those who were present, at the celerity with which it did its work. When it was found that the growth alone was removed and the epiglottis remained intact, the question arose of how this could have been accomplished, for here was the lingual surface exposed as cleanly as if partially dissected off with a scalpel. There are two modes of solution that I would offer to this problem, for I can call it nothing else. One is, that the growth, from the constant efforts of deglutition, had become partly movable upon the epiglottis; and the other, that there was a sulcus or groove at the posterior part, between the growth and the proper edge or margin of the valve. I am disposed to believe in the first, although it is more than probable that the second was a corollary to the first. And what perhaps strengthens this view is that the structure and consistence of the tumour were much softer than that of the cartilage left behind, although some of the latter structure was found to enter into its formation as well.

The general health began to improve, cicatrization was remarkably speedy, the dysphagia disappeared as soon as all tenderness of the raw surface subsided, and, as I had anticipated, there was no contraction, which was proved by the passage of a bougie without obstruction or difficulty. The remaining point for consideration was the ultimate result. Is the nature of the tumour so benign as to lead to the supposition that it will not return? Upon that I dare not venture an opinion, but shall hope for the best. If the surface of the wound has completely shrivelled up by the various applications used, it is probable that an immunity may be the result. At any rate, and under all the circumstances of the case, everything was done which our advancing knowledge of the diseases of the throat suggested.

Appendix.—She continued in fair health, and swallowed tolerably well, until about the middle of April, 1865, when deglutition was attended with coughing and choking, and the voice became weak and throaty. She came to London,

and on the 26th of April I examined her throat, and discovered a fleshy looking growth, the size of a small walnut, springing from the left half and edge of the lingual surface of the epiglottis. The base of attachment was not large, but the tumour gave rise to expectoration of blood and mucus. The right half of the epiglottis, although thickened, had somewhat regained its normal appearance. In addition to the tumour in the throat, another, hard, tense, red, and immovable, had been growing in the left sterno-mastoid muscle, low down, the size of a small hen's egg, having commenced four months previously. A similar one was appearing in a corresponding situation in the opposite muscle. She was pale and thin, and, as she now swallowed very little food, the removal of the growth was determined upon to get rid of the mechanical impediment to deglutition, although now there could be no doubt that the character of the original disease had changed to one of a malignant nature. Its removal was accomplished on the 28th, at 10 a.m., in the presence of Mr. Ure, Mr. W. F. Teevan, Dr. Althaus, and Mr. J. G. Randall, by the same means and appliances as were adopted on the first occasion. With the aid of my left index finger, the thick wire of the écraseur was passed around the growth, and on pulling it home the upper half was cut off. The instrument was reintroduced, and the wire well applied to a groove at its base, and the remainder was snared and cut off, the consistence of the base being much firmer than the upper or spongy part. Very little bleeding followed, and swallowing aud speaking were at once improved, and subsequently continued good. The two pieces of tumour, when placed together, equalled the dimensions of a small walnut. She now gained health and strength, and left London for her home on the 9th of May. On examination of the tumour Dr. Andrew Clark found it to consist partly of an ordinary epithelial growth, but mainly of epithelial cancer. From the characters of the cells, especially their possession of vacuoles and vesicular nuclei, from the disorderly manner in which they were heaped together, and from the absence of every indication of development as distinguished from growth, he was of opinion that,

unless the incision by which it had been removed extended beyond the limits of the growth, it would return either in the cut surface or textures immediately adjacent. Indeed, his written opinion was that it would return under any circumstances; the relief, therefore, afforded by its present and second removal can only be temporary.

ON

VASCULAR PROTRUSION OF THE EYEBALL.

BY

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In the forty-second volume of the 'Medico-Chirurgical Transactions' (for 1859) there is published an account of four cases, in which I tied the common carotid artery for that affection of the eyeball and orbit which, following the opinion of Travers, has since his time been commonly called by the name he gave to it — "Aneurism by Anastomosis of the Orbit."

It may, perhaps, be recollected that attention was first called to this important disease by the publication of a case of it, by Mr. Travers, in the second volume of the Society's 'Transactions;' soon afterwards, Mr. Dalrymple related a case in the sixth volume. By both of these surgeons the disease was considered to be identical in its nature with that condition of the blood-vessels which John Bell had shortly before described, and named "Aneurism by Anastomosis."

For many years no other case, so far as I know, was recorded; and though during the last twenty years several cases have been met with, both in this country and abroad,

the affection must still be regarded as among the rarer forms of disease.

Since the publication of the four cases in the forty-second volume of the 'Transactions,' it has happened to me to have treated three other cases; of these, two were of spontaneous origin, and one the result of injury to the head. I have also had the opportunity of examining the parts immediately implicated in a man and a woman whom I had treated for the affection, and whose deaths occurred long subsequent to deligation of the carotid, from causes entirely unconnected with the operation. I have therefore presumed to hope an account of these cases and post-mortem appearances, may be acceptable to the Society, especially as I believe they not only confirm my former observations, but also conclusively establish the true nature of the affection, which hitherto has been so obscure.

In the twenty-second volume of the 'Transactions' Mr. Busk published an interesting report of a case, exhibiting very similar symptoms to those treated by Travers and Dalrymple, but which was of traumatic origin, supervening upon a violent blow on the head, whilst theirs appeared spontaneously. In this, as in the former cases, the common carotid was tied with success. Mr. Busk considered his case, though arising differently, to be essentially of the same nature as those of Travers and Dalrymple, and remarks—

"I have alluded to these two cases partly with a view of drawing attention to the probability of the supposition I am inclined to adopt, of their being instances of true aneurism in the orbit, and not, as has been considered, of erectile tumours or aneurisms by anastomosis, and in support of this view I would venture to suggest that these cases agree in three essential particulars.

"1st. The sudden accession of the disease, attended with pain.

"2nd. Its rapid increase.

"3rd. The powerful pulsation occurring in the tumours when recent and small—in all of which circumstances do they differ from the erectile tumours described by Mr. John

Bell . . . . the presumption of the affection being in all really an aneurism of some vessel in the orbit appears to me materially strengthened."

These observations of Mr. Busk are so weighty as to show that the affection cannot be aneurism by anastomosis, as for a long time was supposed; and, in the absence of any post-mortem examination of the parts concerned, might, not unnaturally, be thought sufficient to justify the conviction of its being, as Mr. Busk supposed, "a true aneurism within the orbit." This opinion, however, I incline to think is not correct, for, though I am by no means prepared to deny that true aneurism may never occur to any of the intra-orbital arteries, or that possibly some of the reported cases may not have been of this nature, I believe that in several of the cases there has not existed any aneurism at all, and, in the great majority, where there has been aneurism it has been within the cranium, and not in the orbit. Further, that the prominent symptoms are not essentially dependent upon an aneurism, either true or false, whether in the orbit or in the cranium, as a primary or necessary cause, but are, so to speak, secondary to it; indeed, that they may arise from other and varied causes which produce post-ocular pressure upon the ophthalmic veins. For this reason I have ventured to suggest the name of "Vascular Protrusion of the Eyeball," as more in accordance with the pathology of the affection than aneurism of the eye or orbit, which may or may not exist, while I believe pressure, direct or indirect, upon the ophthalmic veins will always be found.

I will, however, briefly give a narrative of the three cases, and of the appearances of the parts in the two post-mortem examinations, and then add such observations upon the nature of the affection as it appears to me these seem to justify when taken in conjunction with the four cases previously reported.

CASE 1.—*Traumatic vascular protrusion of the eyeball; deligation of the carotid; cure.*—The first case was of traumatic origin. Mr. J. H—, a publican, æt. 40, when in a state of

intoxication, was thrown with considerable violence from his horse. He was taken up quite insensible, and so continued for thirty-six hours. For several days he was partially unconscious. There was a free escape of blood from the left ear. Mr. Hepworth, the surgeon who attended him, thinks that considerably more than half a pint of blood escaped during the three or four days it continued to flow. He believes it to have been all blood, and not serum. The accident happened four miles from the patient's home, to which he could not be removed for several days. There was very little external injury. All the symptoms indicated fracture of the base of the skull. Some few days after the accident the left conjunctiva was observed to be congested, which condition gradually increased, the lids became swollen and red, the conjunctiva chemosed, while the pain became very considerable and the sight materially impaired. It was supposed the case was one of acute conjunctivitis, as appears to have occurred in Mr. Curling's (Vol. XXXVII, 'Med.-Chir. Trans.'). It was for this condition that my opinion was first desired, a month after the accident, when I learned the foregoing particulars. The nature of the affection was at once obvious. The eyelids were swollen and dark; they could not be closed over the globe. The conjunctiva was greatly chemosed and purple, with large congested blood-vessels; the globe protruded considerably, and was much congested. The pupil was dilated and inactive, the lens muddy, and vision materially impaired. He complained of a distressing sensation of distension and aching pain in the globe, and there was a decided pulsatory movement of it perceptible to both sight and touch, as well as a very audible sound. There was confusion in the head, with great noise and pulsation in the left ear, the hearing of which was much impaired. All the symptoms were worse in the horizontal position, than in the erect, so much so that he sat up in bed during the night to avoid the distress in lying down, and all were materially checked by pressure on the left carotid artery. He was still ill, and very weak from the accident, and had not fully recovered his memory, so that it was somewhat difficult to determine pre-

cisely how much of his suffering should be attributed to the primary accident and how much to the condition of the eye.

Though, as the case was so acute, and had so rapidly and steadily increased, I had little expectation of any other treatment than deligation of the carotid being of any use, I thought it proper to try. The upright position was enjoined, ice was directed to be constantly applied to the eye and brow, and digitalis, saline purgatives, with sedatives, to be taken. The depression was so great, and the pulse became so weak and irregular, that in four days the digitalis had to be omitted. As at the end of a week all the local symptoms had increased, and the sight had so far gone that there was only a perception of light, while his strength had still further failed, I tied the left common carotid. The room was a dark one, with a very small window, and a violent thunderstorm came on while the operation was in progress, so as to cause a little difficulty in seeing into the wound. Neither vein nor nerve was exposed. On tightening the ligature the confusion and noise in the head and ear instantly ceased, the hearing improved, and the protrusion, pulsation, bruit, and vascularity in the eye, were greatly lessened. Chloroform was not given in this or either of the other cases where I have tied the carotid artery. He went on as well as possible, the sight improved, and the distress was nearly gone, for the first four days; when, as happened in more than one of the other cases I have treated, the eye became so much more vascular and prominent as to cause the patient and his friends to think the disease was returning as badly as ever. After two or three days, however, in him, as in them, these symptoms again subsided, and he steadily and rapidly improved, so that at the end of three months he was well. The eye had completely retired within the orbit, and had recovered its normal aspect, except that the sclerotic remained somewhat darker and duller in colour, as has always happened. The sight had so far recovered that he could read moderate-sized print with facility. The noise in the head had ceased, the hearing of the left ear was restored,

and the memory was good. It is now more than twelve months since the operation, and he continues perfectly well.[1]

CASE 2.—*Case of spontaneous origin, incomplete; treatment refused.*—The second case was of spontaneous origin, but not so acute as most of the others. Mrs. Jane R—, a stout short woman, æt. 47, applied first in May, 1864, at which time the eyeball was decidedly protruded, though it could still be covered with the lids; these, as well as the conjunctiva, were red and tumid; the ball itself was also considerably congested, the iris was dull and sluggish, with the pupil rather dilated, and the sight dim. There was considerable throbbing stretching pain about the orbit, with a pulsating projection of the globe synchronous with the pulse; there was dulness of hearing and noise in the ear of the same side, with confusion and giddiness in the head, all of which symptoms were made worse by stooping or exercise, so that she could only partially attend to her house work. Pressure on the carotid artery lessened all the symptoms, which at once returned on its being removed. She stated that four or five months before, soon after getting out of bed, she was seized with "a giddy fainting fit and something queer in the head," immediately after which the eye began to be affected, and had gradually got worse. I gave her cold lotion with mercurial purgatives. It was arranged that she should be taken into the hospital for more careful watching, but she did not present herself. In August last I saw her at her own home, when she stated she had experienced so much distress from the shaking in her journey to Leeds, and that for some days afterwards the symptoms were so much aggravated, that she had been afraid of undertaking it again, which, however, was not the whole truth, as the subjoined note will show. There was not any very great alteration in the symptoms, but on the whole they had somewhat increased. She promised to attend, but as she did not I communicated with her medical attendant. The following is the reply:—

[1] June, 1865.—The condition of the eye has now become so far normal that it would not be perceived that any thing had been amiss with it without careful examination.

October 24th, 1864.

"I saw Jane R— yesterday; she promised to come to Leeds on next Tuesday. You will find that she is worse. I have wished her repeatedly to see you, but without effect; she was afraid of some operation or other."

The woman had bronchocele.

CASE 3.—*Cancer in the orbit and cavernous sinus; deligation of carotid artery; cure of protrusion of eyeball; subsequent death; necropsy.*—The third case is curious and interesting, as throwing considerable light on the true nature of the protrusions of the eyeball; I watched it for two and a half years. Were I to relate the details of it, and the different speculations from time to time entertained as to its character, I should occupy unnecessarily the attention of the Society; for though at the time of the operation, and for some months afterwards, none of the medical friends to whom I showed the man, any more than myself, could feel sure as to its true pathology, this in the end, even some time before death, became sufficiently manifest, and therefore a few extracts from my notes as the case proceeded will be sufficient. The cast was taken the day before the carotid was tied.

The patient, Joseph Armitage, æt. 43, a stout, thick, broad-set man, employed as cleaner of woollen rags and also as a musician, playing one of the larger brass instruments—the trombone, I think—first applied to me in February, 1862, on account of considerable protrusion of the right eyeball, which he stated had been coming on for four months. It projected about half out of the orbit, and could with difficulty be covered with the lid. Though there was some vascularity of the conjunctiva and sclerotic, this was not excessive; there was some indistinct pulsation; the sight was dim, but not lost. He complained of giddiness and confusion in the head, particularly on stooping, so that he could not do much work. Suspecting there was pressure on the cervical veins from some cause, I examined the neck and found a considerable bronchocele, nearly joining up to which, but still quite unconnected, was a large tumour on the sternum. Of the en-

larged thyroid he appeared to be hardly conscious, and could give no account whatever, except that he had had it a long time, and thought blowing his instrument had something to do with both swellings. There were also two considerably enlarged glands, one on either side of the neck, unconnected with either the bronchocele or sternal tumour.

The sternal enlargement was first noticed about ten years ago, as a small, fixed, hard lump, which had so gradually increased, without pain or inconvenience, that he had hardly taken any notice of it, and thought so little about it that neither he nor his wife, who accompanied him, would have named it had I not discovered it. Both of them, however, were certain it had not increased during the last twelve months, since he had given up blowing the trombone. The tumour was smooth, hard, inelastic, not sensitive; it was altogether without fluctuation, though it communicated most distinctly the sounds and pulsation of the breast. These sounds were unusually loud and distinct; and all the arteries, particularly the carotids, and more especially the right one, appeared to be much larger than common. The bronchocele was largest on the right side, and from its pressure caused the sterno-cleido muscle to appear broad. There was some roughness in the respiratory sounds from pressure on the trachea. I saw the man frequently during the year up to February, 1863, during which the sternal tumour did not increase; indeed, both he and his wife thought it had decreased. The bronchocele had steadily increased, particularly downwards, so as nearly to reach the sternum. The two cervical glands had varied in size, but on the whole had increased. The eyeball had steadily more and more projected until it protruded altogether beyond the orbit, pushing and stretching the lids, particularly the upper one, before it; not unfrequently this slipped back and left the globe uncovered. The lens was clear, but the pupil was dilated, and vision so far gone that he was doubtful of the position of the window. The ball was more congested than formerly, but still not in an excessive degree. The orbit was filled with a soft pulsating mass, into which the globe could be partially pressed

back. Pressure on the right carotid stopped the pulsation and lessened the protrusion of the ball, both of which returned on the pressure being removed.

During this period two other tumours had appeared. One at the outer side of the orbit, just above the zygoma; it was soft, compressible, and pulsating; by pressure it could be much lessened in size. If pressure were maintained on the carotid the tumour very slowly enlarged, but rapidly if the vessel were free. It apparently communicated with the orbit, as pressure upon the tumour caused the eye to be more prominent and pulsating, while pressure on the ball rendered the tumour fuller and harder. On the right side of the head, near the centre of the parietal bone, a second, soft, elastic, compressible, pulsating tumour, of the size and shape of half a small orange had formed. It could be considerably lessened by pressure, on removal of which it slowly rose again, with a distensible movement. To it, and over it, passed large and freely pulsating arteries from the posterior temporal. Neither of these tumours was tender to the touch, but the general distress was so great, and the pain from the eye so tormenting, that he urgently desired something should be done. Many speculations were started as to the precise nature of the affection, none of which, however, carried conviction. But as it appeared certain that the protrusion of the eyeball was due to some posterior, if not intracranial, pressure, which was distinctly relieved by closure of the carotid artery; as vision was lost, and sloughing of the exposed eyeball impending; as these distressing symptoms had materially increased during the past month, and, still more recently, deafness in the right ear had supervened, with great confusion from the constant noise over the right side of the head, and believing the cause of the protrusion of the eyeball to be essentially of the same character as those where I had successfully tied the carotid artery, I thought the predominant distressing symptoms might be lessened by tying this vessel; while, should the sternal tumour prove to be, as some men of great experience thought it not improbably might be, aneurism of the arch

of the aorta or innominata (a view in which I did not participate), no harm would be done, as it would be only arresting the blood partially on the distal side.

On February 17th, 1863, I placed a ligature round the carotid artery.

Owing to the large thyroid gland and the many considerable arteries and veins which I was anxious to avoid, the operation was not an easy one. One large vein passed so directly across the middle of the incision that it could not be avoided; both ends were tied with fine flexible iron wire. I had intended to have tied the ligature below the omo-hyoideus, but this muscle was spread out to such a breadth by the bronchocele that it had to be placed above, or the muscle must have been divided. Neither vein nor nerve was seen. On tightening the ligature all noise and confusion in the head instantly ceased; the eyeball became less prominent; the two swellings lessened in size, and ceased to pulsate.

For two days the man was as well as possible, when the wound became erysipelatous, with a very severe accession of general febrile symptoms (the day of the operation a man residing at the adjoining house had been seized with violent cellular erysipelas), which for thirty-six hours placed his life in considerable danger, after which the wound freely suppurated, and he greatly improved. On the morning of the 21st (the fifth day), after a very good night's rest, the left side of the body was found to be partially paralysed. This during the day increased to be one of the most complete attacks of hemiplegia I ever saw, as there was not the faintest power of motion or sensation in any part of the side, notwithstanding which both the local and general symptoms improved. On the 25th he became very restless "from pain in the left leg." On the 26th he could just stir the toes. On the 28th (twelfth day) the ligature was lying loose in the wound, which soon after closed, but opened again, and then continued to suppurate, and remained hard for some time. The leg rapidly regained power and sensation. On March 26th he could draw it up, and could just move the fingers a little. Towards the end of May he had recovered sufficient

power on the side to be able to sit up for two hours in the day, and, indeed, the left leg had nearly as much power as the right, but the arm had not recovered in the same ratio. During this period he had been two or three times nearly carried off from diarrhœa, scanty bloody urine, and a distressing cough, with bloody expectoration. Soon after the operation the eyelids had left the ball so completely uncovered that more than once I passed a wire suture through them to retain them over it, but without much success. The cornea became opaque and the ball soft. It gradually atrophied, without suppurating.

The orbital mass had gradually lessened, and there was no pulsation in it or in the cranial tumours. These, which at first were lessened, were now about as large as before the operation.

The sternal tumour continued unaltered; the left carotid artery in its whole course, and the right up to the ligatured spot, pulsated vigorously with enormous volume. The veins of the neck were very large, particularly the right external jugular, in which there was a free pulsatile motion. The voice and respiration were rough, and there was a troublesome irritable cough, with a free expectoration of bloody mucus.

In the middle of June he had sufficiently recovered to return home, ten miles from Leeds.

In the middle of August he came over to see me. My notes are—"He has been in tolerable health, except having had two or three attacks of diarrhœa, which for the time reduced him. He has lost flesh, but is considerably stronger, and can walk up a stiff hill a mile long—fully equal to two miles on level ground. The left arm has not recovered so much power as the leg, though he can use it for most purposes. The bronchocele and sternal tumours are unaltered. The action of the heart is less tumultuous, and the arterial volume diminished. The wound in the neck is perfectly sound, and there is less distension and pulsation in the carotid below where it was tied. The eyeball is reduced to a mere speck, level with the cheek. The lids are natural; the orbit is filled with solid matter, but which is influenced by

pressure on the zygomatic tumour, as the latter is by pressure on the orbital mass. This side tumour is, I think, somewhat larger than it was two months ago; it is soft, compressible, and communicates with the orbit, as a finger placed upon each shows. There is not the least pulsation in it, or in that on the head. This latter has not much altered; perhaps it is more solid. There is neither noise nor confusion in the head. The kidneys now secrete well."

He went on with little alteration until the middle of December, 1863, when his wife called to say that he had suddenly lost the use of the right arm. That a few days before, while making a sudden snatch at one of his children, who had offended him (but who escaped the clutch) the arm fell useless. Suspecting fracture, I sent my son, Mr. J. A. Nunneley, to see him. He found the humerus broken in the middle of its shaft, and put it up with splints. He reported that the man was not much altered, except that he found a considerable tumour over and involving the left scapula, which had been growing rapidly since he had been seen.

The patient was visited occasionally by my son, and I heard of him from his wife every week. The humerus united well. He lingered on, getting weaker, with the sternal and scapular tumours growing larger, until the middle of August, 1864, when he sunk exhausted by diarrhœa.

Necropsy.—I made a post-mortem examination two days after his death.

The body was greatly emaciated. The nature of the disease was now obvious enough; there was an enormous tumour under and partly involving the left scapula. The sternal tumour was much larger, and in the skin all around it were scores of small tubercles; it reached up to the enlarged thyroid gland, and apparently was confounded with it. The glandular swellings in the neck were much larger; the right orbit was filled with a tuberculous mass, which also projected from it; the zygomatic and parietal tumours were of about the same size as they had been for a long time. No other similar tumour had appeared on the right side of the head.

The skin had not given way in any place. The right humerus was again fractured, and the bone much splintered. It had given way with an audible crack two days before his death, while his son was turning him over in bed.

Before section of the body malignant disease was obvious enough. The sternal tumour had apparently commenced in the bone, the texture of which had nearly disappeared. The mass projected as much into the chest as it did externally, pressing the lungs upwards and the heart downwards to the left side. The lungs were not diseased; the heart was large and soft, but not otherwise abnormal, it was partly filled, particularly the left ventricle, with clear tenacious fibrine; the thyroid gland was one large mass of medullary cancer, which pressed upon the trachea and œsophagus. The zygomatic and orbital tumours were one, communicating through a large opening in the wing of the sphenoid bone. In the orbit the mass was firmer, but at the zygoma it was like thick porridge, thus accounting for the lessening of this tumour and the retiring of the eyeball as either of them was pressed on. The parietal tumour passed through a large opening in the bone into the skull upon the dura mater, to which it was attached. It was very soft, like the other tumour, thus accounting for its being lessened on pressure, and the pulsation. On opening the skull the surface of the brain was found covered with serum; its structure was soft, but otherwise normal. By the side of the sella turcica, passing through an opening into the apex of the right orbit, *was a continuation of the diseased orbital mass which had obliterated the right cavernous sinus. This mass was more solid than those on the head. The ophthalmic vein had been pressed on until it was lost in the mass,* thus, in my judgment, proving that the ocular protrusion was essentially of the same nature in this as in the other cases I have reported, different as the material causing the pressure might be. The vessels constituting the circle of Willis were alike on both sides. The right carotid had been securely tied. Below the ligature and the left carotid in its whole course the vessels were at least double their normal size, and would just admit the tip of the little finger.

The abdominal viscera appeared healthy, except the liver, which was dark and soft, but not affected with malignant disease.

Necropsy of Case 4, related at page 187, *Vol. XLII, 'Med.-Chir. Trans.'*—The other post-mortem examination which I have had the opportunity of making is the fourth case mentioned in the paper referred to. The disease began instantaneously on the 28th of July, 1859. The carotid was tied on the 21st of August following. The woman perfectly recovered, though some considerable time elapsed before the globe altogether retired into its normal position in the orbit and the swelling and congestion of the lids had entirely passed away. However, in the end they did, and all the parts resumed their natural appearance, except the lens, which remained opaque, and the sclerotic a little dull. I had frequent opportunities of seeing the woman, who always said she was well, except that she had some difficulty of breathing, which I attributed to the large bronchocele. After her recovery from the operation she continued to perform the household work of the family until the end of February, 1864, though latterly she had complained of being weak and lethargic. On the 26th of February she was seen by a medical man, who found her quite sensible, but suffering from what he thought to be a severe attack of bronchitis, which was then very prevalent. In the afternoon of the following day symptoms of serous apoplexy supervened, and she died the same evening.

I examined the head and neck forty hours after death. The veins of the scalp and all the cerebral sinuses were full of black fluid blood. The arachnoid membrane was thicker and more opaque than natural. All the vessels of the pia mater were congested. The brain was small, with some clear serum upon its surface. The upper surface of both cerebral hemispheres was alike; but on cutting through these, on a level with the corpus callosum, the whole of the anterior portion of the right hemisphere, particularly the fibrous part, was found to be considerably less than that of the left side. This was

most marked on the under surface, so that the inferior part of the right anterior lobe was not much more than half the size of the left. There was no other change to be noticed in it; the vascularity, colour, and firmness of the two sides were alike; the blood-vessels of the two sides of the brain were equally patulous, and of like size.

On the right side of the sella turcica was found a circumscribed aneurism of the ophthalmic artery, just at its origin, as large as a hazel-nut, which was filled with a dense, solid, red clot, so loosely attached to the arterial walls that when these were cut into the clot at once fell out. *The ophthalmic artery continued forwards into the orbit, but both trunk and branches were of very small size.*

The right carotid divided, as usual, into its three branches, which were of equal size with those of the left artery, and the circle of Willis was normal.

How far the diminution in size of the right hemisphere was dependent upon the closure of the carotid of the same side it is difficult to determine. At first sight, it appears reasonable to suppose that they might stand in the relation of cause and effect, but this, I would observe, appears to me to be, to say the least, doubtful, as the two sides of the brain appeared to be equally full of blood, and all the arteries were equally patulous; besides, if the cutting off of the supply of blood was the cause, we should have expected the effect to have been produced immediately, or shortly after the ligature had been applied, as happened in the man whose case is above related; in the woman who died after the operation, referred to in Vol. XLII; and in cases reported by other surgeons, after ligature of the carotid for various affections.

Mr. Busk, as we have seen, had called his "A Case of Aneurismal Tumour in the Orbit,"[1] and Mr. Curling his "A Case of Traumatic Aneurism of the Ophthalmic Artery,"[2] and thus showed their conviction of the nature of the affection as seen by them supervening upon injury to the

[1] 'Med.-Chir. Trans.,' Vol. XXII. [2] Vol. XXXVII, id.

head. Yet, as no post-mortem examination of the parts had been made, the proof was still wanting to overturn the idea of aneurism by anastomosis originated by Travers, and so long entertained by the profession. In the paper referred to I gave several reasons, as well as the result of the examination of the parts in the one case which had died after the application of the ligature, which I thought to be conclusive against the long-received opinion of the nature of the affection; but the history of the case of Armitage, now given, the condition of the parts concerned found on death, taking place eighteen months after the carotid had been tied, and the post-mortem examination of the head of Mrs. Jackson, nearly five years after the operation and cure of the protrusion, so decisively show that the idea of such protrusion of the eyeball being aneurism by anastomosis in the orbit is altogether erroneous, that there cannot now be any hesitation in abandoning it. *In fact, in the great majority of such cases of protrusion of the eyeball there is no disease whatever in the orbit; the seat of it is most commonly intra-cranial. The protrusion of the eyeball is passive, and the other distressing symptoms are secondary, depending upon obstruction to the return of the blood through the ophthalmic vein, just as happens in those cases of popliteal and axillary aneurisms where the limb swells below the tumour, because this presses upon the accompanying vein.* That this pressure, in the great majority of acute spontaneous cases, is caused by an aneurism of the carotid as it emerges into the cranium, or of the ophthalmic artery near to its origin, is, I think, now certainly proved. In cases of traumatic origin, supervening soon after severe injury to the head, the cause is most probably effused blood near to or within the cavernous sinus, while in those cases where the protrusion does not come on until some time after the receipt of the injury, or where the violence has not been so considerable, it may be serum or fibrine, or even pus, the result of suppuration in the sinus. This latter was most probably the true cause of the protrusion of the orbital contents in the woman who died after the diligation of the carotid in Moorfields Ophthalmic Hospital, where the protrusion

had not supervened until some time after the receipt of a violent blow with the fist upon the temple, and where, as only pus was found after death in the cavernous sinus, some doubt appears to have been entertained as to the correctness of the diagnosis and the propriety of the operation.

That the aneurism of the ophthalmic artery, or effused fluid, whatever its character, or even a tumour, may be seated within the orbit rather than in the cranium, is quite possible; but inasmuch as those conditions which would make a small amount of pressure felt considerably do not exist in the orbit, but do exist to a very considerable degree immediately behind its apex in the cavernous sinus, it will, I apprehend, be in reality found that in this situation the actual cause does exist in the great majority of cases. In the orbit there is a large quantity of soft, yielding material, which would require a body of considerable size to cause sufficient pressure to produce the urgent symptoms witnessed in a sudden and acute case of protruded eyeball, whether of spontaneous or traumatic origin; whereas, bound closely together as the artery, veins, nerves and absorbents, are in the dense, unyielding fibres forming the walls of the cavernous sinus, a very small cause will necessarily produce a very great effect. Further, it must not be forgotten, that it is very rare to meet with aneurism in any of the arteries of a size such as are found in the orbit. When an aneurism does spontaneously form, it is almost invariably found in connection with a vessel of some considerable size, the coats of which are far more obnoxious to that loss of elasticity which predisposes the inner one to give way than are those of the size of the orbital branches. The ophthalmic artery is much larger at its origin and in the cavernous sinus than it is soon after entering the orbit.

When the protrusion takes place more slowly, and is unaccompanied by the acute symptoms usually resulting from aneurismal pressure, we may, not unreasonably, especially if there be any corroborating indications, as in the case of Armitage, suspect the cause to be some growth in or near the cavernous sinus, or some tumour or effusion within the

orbit, and near its apex, rather than aneurism, whether true or false.

I would further remark that, though this vascular protrusion of the eyeball in its most acute form is evidently one of the rarer diseases, or more frequent instances of it would have been recorded (for I cannot suppose that the fact of so many as seven cases having been treated by myself within the space of twelve years is more than one of those curious fortuitous circumstances for which it is impossible on any rational ground to account), *I believe in a less intense degree, and more chronic condition, protrusion of the eyeball resulting from posterior venous congestion is by no means uncommon. Whatever obstructs the return of blood may indirectly, if not directly, induce fulness of the eyeball. Tumours of every kind, in the orbit, cranium, or neck, may be all causes.*

Enlarged thyroid gland is certainly a very frequent cause. In at least five of the cases of spontaneous origin which I have seen the thyroid gland was of large size; and, although in two of these the cause was the sudden formation of aneurism in the cavernous sinus, and in another the deposit of cancerous matter, as proved by the post-mortem examinations, we cannot trace any connection between the enlarged gland and the weakened arterial coats, yet the coincidence is worth notice.[1]

Anæmic young women with bronchocele very frequently have marked fulness of the eyes, the protrusion of which occasionally goes on to such an extent as to cause serious anxiety and inconvenience. This is a state which has long been known, and the obscurity of its origin and the difficulty of its removal commented upon. Lately, some most excellent observers, in this country as well as on the Conti-

[1] It may also be mentioned that the sister of Mrs. Jackson has a large bronchocele, and that I have the mother under care for one of the largest bronchoceles I ever saw, which will probably, ere long, kill her, as the skin has ulcerated, and a large fungoid mass of granulations (non-malignant) bleed, slough, and form again, without there being the least disposition to cicatrization; also that Mrs. R— states her mother had an enormous bronchocele.

nent, have written upon it, and noted the coincidence of ocular protrusion and bronchocele. In goïtre it is often most marked.

The glazed and prominent eye is known to everybody as a very usual occurrence when there is a feeble, flabby, dilated heart; particularly when the valves of the right side act imperfectly. In emphysema and chronic bronchitis, and confirmed asthma, nothing is more frequently seen than protrusion of the eyeballs. In fact, whatever obstructs the return of blood to the right side of the heart, or its escape from the cavities of it, is in the end almost always accompanied by, if it does not directly cause, protrusion of the eyeballs. In many of these cases it may not improbably happen that the impediment to the return of the blood is accompanied, and the protrusion made worse, by some effusion into the loose post globular tissues of the orbit; for nothing more surely tends to cause such exudation from the weakened vessels than impediment to the return flow of the venous blood. Thus this effect becomes in its turn an aggravating cause. In the weak anæmic young female, where the blood is wanting in its due proportion of fibrin and globules, a very slight impediment may be sufficient to cause exosmosis of the thin serum. A slight pressure on the venous stream, possibly only a functional disturbance, or a cause so slight as to be overlooked may be all, and thus the well-marked prominent symptom of protrusion of the eyeballs be regarded as the disease itself; whereas it is only the effect of a more obscure but more important abnormal condition. It is, I think, certain that such a state never supervenes in the robust and strong unless there is evident pressure in the course of the veins.

At the present time I am attending two ladies, both above the middle age, in each of whom, I have little doubt, considerable prominence of one eyeball results from some vascular fulness or effusion in or behind the orbit.

In one, a pale, weak, and very nervous lady, who, since girlhood has had enlarged thyroid gland, both eyes are prominent; but the right one has, since a severe attack of bron-

chitis with persistent cough, become so decidedly more so, with pain and congestion, as to excite alarm, and to be mistaken for conjunctivitis and treated accordingly. In the other lady the attack is more acute, and was very sudden. I suspect here there is a small clot of blood pressing upon the ophthalmic vein. She was for two years in declining health; and had numbness of the arm and bleeding of the nose. The attack came on instantaneously whilst sitting out of doors on a cool evening, after travelling in an open carriage exposed to the sun on a very hot day on the continent. The pain was so great and the protrusion of the eyeball so considerable, that it was mistaken for general ophthalmia, and treated with leeches, mercury, and atropine. I doubt not many members of the Society can recall similar instances which have fallen under their notice.[1]

Appendix.—Since the reading of this paper Dr. T. G. Morton, of the Pennsylvania Hospital, has related a case of spontaneous origin, in which he has recently tied the carotid artery with success.[2] In the comments appended to this case, Dr. Morton has alluded to the abstract of this communication as published in the Journals, and has adduced two statements which he thinks conclusive against the opinions I have advanced as to the seat of the disease being rather post- than intra-orbital—viz. "in many cases distinct and readily defined aneurismal tumours could be felt in the orbit," and that in "severe cases cures have been effected by injecting coagulating fluids into the aneurismal sac." These circumstances, Dr. Morton considers, could not happen were there not a true aneurism within the orbit itself. I must confess these two arguments do not appear to me to possess much value, for who does not know that an aneu-

[1] July, 1865.—Under the influence of iron, iodine, and strychnia, gentle irritants behind the ear, good diet, and change of air, both of these cases are so much improved that they may be said to be cured, though, on careful observation, a slight prominence in each eye may still be perceived.

[2] "On Orbital Aneurisms," 'American Journal of the Medical Sciences,' April, 1865, page 318.

rismal anastomosis, even of large size, may not unfrequently be cured by the injection of a coagulating fluid into it? and in precisely the same way would such fluid act upon the congested and distended blood-vessels in the orbit, if this condition be caused in the way I have supposed it to be in these cases. By the coagulation of the blood in them, their occlusion and the subsequent absorption of the coagulum, the distending force would be removed. But I apprehend Dr. Morton would not, as a rule, expect such treatment to be successful in either one form of the disease or the other. As to the existence of an aneurismal sac being felt in the orbit, I think he must be a very bold man, having far more or much less experience than most surgeons, and possess far more than the average amount of the "*tactus eruditus*," who would unhesitatingly declare the existence of an aneurismal sac from tactile sensation alone, in such a situation, with such symptoms as actually exist in these cases. When he recollects how difficult the wisest and best men find it, in many cases, to diagnose by touch between many deep-seated swellings, and particularly when he is informed that it has actually been proved, in the only four cases where post-mortem examinations have been made, *no tumour, aneurismal or otherwise, has existed within the orbit*, I do not see how the assertion can be clung to. We cannot rely too much upon Mr. Guthrie's case, where the symptoms during life are not very distinctly set forth. In the case of traumatic origin operated upon in the Moorfields Ophthalmic Hospital, which Dr. Morton has himself referred to as so evident as not "allowing the slightest doubt as to the nature of the affection," all "the arteries were found quite healthy," and the only disease was in the post-ocular veins; and in the two cases of spontaneous origin, of which I have related the post-mortem appearances, during life the pulsating protrusion of the orbital contents was so decided that they might have been taken as illustrative cases for the existence of aneurismal sacs, yet after death no disease whatever was found in the orbit, whereas in the cavernous sinus it was evident enough.

Of the treatment to be adopted I would only venture to say that in the most acute attack, whether spontaneous or traumatic, I believe tying the common carotid of the same side will be found the best. Indeed the symptoms are so urgent as to necessitate the promptest measures, or the eyeball and lids will slough. Of the six cases in which I have adopted the plan, five were cured, and in the one which died the affection of the eye was arrested, death resulting from exceptional causes; while in the one case which is still under observation, although by no means so acute as most of the others, where only an expectant plan has been hitherto adopted, the disease appears to be steadily advancing, and in one of those where ligature of the carotid speedily cured the patient, rest and other means had previously been perseveringly but unsuccessfully tried for upwards of seven months. No one would think of placing a ligature round the carotid in the less severe forms, as probably the cause might be removed by treatment, for the pressure may be caused by tumour or effusion, which may be taken away; and in those more frequent and subdued forms, resulting from bronchocele, anæmia, or obstruction in the heart or lungs, alone or combined, treatment must rather be directed to relieving these conditions than to that of the eye, which will be restored to its normal state when the causes of protrusion are removed.

I am aware that two cases of cure by compression of the carotid artery have been reported, one in Padua and one in Verona, and the plan has found some supporters in this country who have tried it more than once; but though pressure appears to have been effectually and vigorously kept up, it has, so far as I am aware, failed to effect a cure; nor should I expect it in the majority of cases to be successful. The common carotid, I apprehend, is one of the least promising of the large arteries of the body to subject to compression; should a clot form in the vessel and become detached it would readily be conveyed to the brain, and there producing embolism, cause considerable danger to life. No pressure can be effectually made upon the artery without

pressing unduly upon the jugular vein, pneumogastric and sympathetic nerves, which, for many reasons, it is desirable to avoid. That a cure may possibly be effected by pressure the two cases prove, and therefore it may be tried. Nevertheless, knowing how almost universally successful deligation of the common carotid artery has been, and that compression has frequently failed, so far as at present adopted, the former plan is the one which I should be prepared to adopt in any other case.

The propriety of active interference is also confirmed by the present condition of Mrs. Jane R— (Case 2, page 20), whom I have lately seen, August, 1865. Though in her the affection has been from the first the least active of any of the cases I have described, the distress the woman suffers is, and has been, very considerable. She is altogether unfit for any work. She has been confined to her bed for upwards of four out of the last twelve months. Even now she can only move slowly about, as all exercise, particularly stooping, occasions giddiness and acute pain in the head. She sleeps badly from pain and noise in the head. All useful vision is lost, yet light is very painful, and induces a sensation of nausea. She keeps the eye constantly covered with a wet cloth, which, she says, lessens the distress. The globe has somewhat receded into the orbit since last August, and there is now less chemosis of the conjunctiva than then existed; but there are several largely congested vessels in it as well as in the sclerotic. The iris is dull, the pupil inactive, the lens is muddy, and there is ptosis of the upper lid. This is a state which contrasts unfavorably with the more acute cases in which the carotid artery was tied.

CASE

OF

ULCER OF THE LOWER PORTION OF THE ILEUM

COMMUNICATING WITH THE BLADDER.

BY

JOHN MORGAN, F.R.C.S.

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Cases of ulcer of the intestinal tube communicating with the bladder are sufficiently rare to justify me in recording the following case, in which an opening from the terminal portion of the ileum into the bladder existed for nearly seven months, and through which the whole of the fæces was passed for the last three months of life.

S. G. B., æt. 60, had spent many years in tropical climates, but had never had any severe attack of dysentery. In March, 1862, he first complained of some pain and slight fulness in the left groin, which soon increased, and a tumour in the left iliac fossa was perceptible. It was hard, lobulated, and somewhat tender to the touch; it gradually increased in size, making way towards the mesial line of the body, when it began to cause considerable irritation of the bladder, with frequent desire to pass water. The tumour, having increased

to the size of an ostrich's egg, occupied the upper part of the left iliac and lower portion of the lumbar regions. It seemed attached posteriorly, and was not movable. The patient had not until this time suffered much in general health, except being somewhat more than usually liable to attacks of dyspepsia and the bowels did not act with their ordinary regularity.

In April, 1863, whilst at Brighton, he was one day much alarmed by the passage of flatus through the urethra. After which the pain in the bladder, and on making water, was so intolerable that Mr. Furner was obliged to order him large doses of morphia and other sedatives. A few hours after the flatus began to pass some feculent matter became mixed with the urine, and the deposit of mucus, combined as it was with shreds of lymph, in this excretion, showed the inflammatory action which had been set up in the bladder by the proximity of the tumour and the presence of fæces in its cavity.

Day by day the urine continued to be mixed with semi-fluid fæces, the bladder by degrees becoming accustomed to the novel character of its contents, ceased to pour out such large quantities of mucus, though he always had to relieve it about every hour. The size of the tumour was not apparently influenced by the discharge; at times it was much more painful than at others. The quantity of fæces passed per anum gradually diminished, while that voided through the urethra increased, and for the last three months, notwithstanding the regular use of enemata, only a few scybalous lumps were passed through the proper channel.

During the progress of the disease, and from the constant distress which this state of things occasioned, as well as from the semi-fluid diet to which the patient found it best to restrict himself, he had become from a large and corpulent man, greatly emaciated, though his countenance never assumed the cachectic aspect so indicative of the presence of malignant disease. About the middle of August a more than usually profuse discharge of fæces occurred, being far more than could be accounted for by the ingesta, this produced great

exhaustion and pain, so that he was compelled to take large doses of opium with stimulants. The abdomen became extremely tympanitic, but on examining the site of the tumour it was evidently greatly reduced in size. From this time his general powers began rapidly to decline, and having been comatose for three days he finally sank in the beginning of October.

Post-mortem examination twenty-six hours after death.— The abdomen was still thickly coated with fat. On opening its cavity all trace of the tumour had disappeared; from five to six inches of the lower part of the ileum were found much distended, the calibre of this portion of intestine being as large as the colon under ordinary circumstances. The coats of the bowel were attenuated and in almost a gangrenous condition, it was adherent to the inner wall of the abdominal parietes, to the upper surface of the pubes, and on dissecting away these adhesions, and laying open the intestine, an ulcer the size of a sixpence, with ragged edges, was found communicating directly with the cavity of the bladder. The intestine then contracted to its natural size, and after one small convolution opened into the ileo-cæcal valve, which, with the colon, was in a natural condition. The other portions of the intestinal tube were perfectly healthy, and free from any appearance of ulceration. The bladder was much contracted, its coats thickened, the mucous membrane rugous and very dark in colour.

During the long progress of the foregoing case the patient saw many eminent members of the profession, but, from a dislike on his part to consultations, I very seldom had the advantage of discussing the case with any of them. The general impression in their minds was, I believe, that the tumour was of a malignant nature.

The chief indication as to treatment was to avoid any food that was likely to irritate the bladder, and he found most comfort from taking everything of a fluid or semi-fluid kind :— soup, milk with rusks, pounded meat or game in the form of panada, constituted his diet, and for some months he took

opiates in proportion to his need. Great comfort was derived from occasionally syringing the bladder with tepid water, but it was remarkable how soon this viscus became reconciled to the constant transmission of fæces through it, which, though at first it caused the greatest agony and severe inflammatory action, was afterwards passed with a comparatively slight amount of distress.

Cases somewhat similar to the foregoing are reported by Mr. Charles Hawkins, Vols. XLI and XLII, 'Med.-Chir. Trans.;' Mr. Sidney Jones, Vol. X, 'Pathological Society's Transactions;' Mr. Mc.Whinnie, 'Medical Times and Gazette,' 1863, page 28.

REMARKS UPON OSTEO-MYELITIS

CONSEQUENT ON

GUNSHOT-WOUNDS OF THE UPPER AND LOWER EXTREMITIES,

AND ESPECIALLY UPON THE

TREATMENT OF STUMPS AFFECTED WITH OSTEO-MYELITIS AFTER AMPUTATION NECESSITATED BY SUCH INJURIES.

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Considerable attention has been given during the last few years, especially in France, to one of the special consequences of gunshot wounds of bones, of considerable interest to military surgeons. I refer to the inflammation which is set up by these injuries in the medullary membrane, that fine vascular tissue which lines and fills the central canals of long bones, and which is continued from them into the cells of the cancelli and the Haversian canals. As is well known, it is by means of the vessels ramifying upon the meshes of this connective tissue that not only the oily substance called

the medulla, which the tissue encloses in all directions, is secreted; but also, in conjunction with the vessels of the periosteal investment, that those changes which are required for the proper nutrition of bone are effected. The tissue which lines the cells of the flat and irregular bones and which corresponds in function with that of the long bones, though modified in certain respects to suit the special characters of the former, appears to be equally subject to the inflammation to which I refer under similar circumstances; but it is to it, as it occurs in long bones, that the attention of surgeons has been particularly directed of late, because it is in them that it has assumed a character of importance in respect to the practice to be adopted in the after treatment of gunshot fractures, especially when the diseased condition of the bone has so far advanced as to lead to the necessity of the removal of the limb or stump involved in the disease, by amputation in its continuity or by exarticulation.

It is not believed that any essential difference exists between the nature of the inflammation of the medullary tissue when developed by gunshot wounds, and the inflammation which occasionally arises after the ordinary injuries and amputations of civil life; but it is its comparative frequency after gunshot injuries and after amputations consequent upon them, in men of previously sound constitutions in military practice, contrasted with the comparative rarity of its occurrence in patients of sound constitution in civil practice, that has caused attention to be so much directed to it and to its proper treatment by army surgeons.

The name *Osteo-Myelitis* has been employed by French and English surgeons for the purpose of defining this inflammatory action and the particular direction in which its influence is mainly exerted. The term *Endosteitis*, used by some German writers (derived from "Endosteum," ενδον, within, and οστεον, a bone, a designation which we occasionally find used synonymously with our term "medullary membrane") seems to express better the special diseased condition intended to be signified, for it serves to mark more clearly the distinction between the endostitic inflammation

and that of ostitis and periostitis. Both terms are used indiscriminately in the course of this paper.

The subject of inflammation of the medullary tissue of long bones after gunshot wounds, and amputations resulting from them, has been specially investigated by several surgeons; but a French military surgeon who was in charge of one of the large hospitals at Constantinople during the Crimean war, Dr. Valette, and M. Jules Roux, the principal surgeon at the large marine hospital of St. Maudrie, at Toulon, have more particularly drawn attention to the subject.

Dr. Valette published a report[1] on the effects of osteomyelitis among the wounded from the battles of the Alma and Inkermann who were received in the hospitals at Constantinople. An analysis of this memoir was laid before the Surgical Society of Paris. On studying this report the reader can scarcely help concluding that the so-called osteomyelitis, in a large number of the instances referred to by Dr. Valette, must have been truly cases of pyæmic poisoning, and that, in all, the symptoms of the osteo-myelitis were greatly aggravated by circumstances tending to the development of pyæmia. Dr. Valette acknowledged the close association between the osteo-myelitis in these cases and pyæmia, and stated that purulent infection was a constant consequence of it; but he regarded it as a far more fatal disease than pyæmia, which occasionally was recovered from, while osteo-myelitis, he asserted, had killed every one who had been attacked by it. The local morbid changes produced by the disease were found by Dr. Valette to vary according to two stages: in the first stage, the medulla was observed to be intensely vascular and increased in density; in the second stage, there was purulent infiltration of the open tissue, with small deposits of pus in the medullary canal. Associated with the latter were generally found the usual signs of purulent absorption, as

[1] Valette Tharsile, Médecin-Major, "Mémoire sur l'ostéo-myélite des amputés," 'Mém. de Médecine, de Chirurgie, et de Pharmacie Militaires,' 2me série, t. xvi, p. 230.

ecchymosis, abscesses in the lungs or liver, or deposits of pus in the joints, muscles, and other tissues. In a few cases no decidedly morbid changes were detected. The following is Dr. Valette's description of the local indications of the disease when occurring in a stump after amputation:

1st.—Protrusion of the medulla from the end of the divided medullary canal in the form of a grayish-coloured button-like protuberance; 2nd, separation of the periosteum from the circumference of the lower portion of the divided bone; and 3rd, protrusion of the bone between the lips of the wound.

The disease in its acute form generally commenced from the fifth to the tenth day after the operative interference, being ushered in by rigors and fever, perhaps nausea, vomiting, and typhoid symptoms; delirium supervened, the skin became icteroid; and the general symptoms which followed, and immediately preceded death, closely resembled those of purulent absorption. Death sometimes occurred rapidly, perhaps on the third or fourth day after the first appearance of the symptoms. Dr. Valette found all treatment ineffectual, such as the application of caustic substances or the actual cautery to the end of the bone, or its removal by the saw; and he was consequently induced to advocate the same view, with regard to this acute form, that M. Jules Roux has since advocated with regard to the disease in its more slowly developed chronic form. He thought that all resections and amputations for excessive osteo-myelitis after gunshot fractures should be abandoned, and disarticulations substituted; the wounded at the same time being scattered in tents as widely as practicable.

The observations of M. Jules Roux refer to osteo-myelitis in a more chronic form. M. Roux had under his care a very large number of soldiers who were wounded in the Italian campaign of 1859, about 2000, and a considerable number among these men presented diseased conditions demanding consecutive amputation, or other surgical interference, on account of the injuries they had received. In consequence of the large ratio of fatal results in the first eight amputations and resections performed, viz. six deaths out of the eight

cases, M. Roux was induced to try disarticulation of the diseased limb instead of its partial removal by amputation or resection, and this change in practice was attended with remarkable success.

The following is a summary of the arguments which led M. Roux to substitute the operation mentioned for amputation.[1] Osteo-myelitis, he says, of a more or less grave character invariably follows all gunshot wounds of bones, especially when the spongy tissue of broad and short bones such as the calcaneum, or the articular ends and medullary canals of long bones, are wounded; and the inflammation of the medulla ordinarily spreads, with more or less rapidity, throughout the entire bone. This osteo-myelitis in most cases yields to rest and treatment, and is cured; but if it assumes a chronic form, and removal of the limb becomes necessary, then secondary amputation generally only takes away a portion of the inflamed bone, and, in consequence of this incompleteness in the operation, the disease is aggravated in the remainder.[2] Hence

[1] 'Bulletin de l'Académie Impériale de Médecine,' 24 Avril, 1860.

[2] Some time after this paper was written, I received from Dr. Fayrer, Professor of Surgery in the Medical College of Calcutta, a pamphlet on 'Osteo-myelitis,' illustrated with histories of thirty-five cases, chiefly occurring in natives of India. This experienced surgeon, both in military as well as civil practice, has arrived at views regarding the treatment of osteo-myelitis closely resembling those of M. Roux. At page 5 of the pamphlet Professor Fayrer writes as follows:—"M. Roux has especially called attention to the subject of osteo-myelitis in connection with gunshot fractures, and the question of amputating above the joint, instead of through the injured bone. He recommends that disarticulation should be performed rather than amputation through the bone. His experience was obtained during the late French-Italian war. My own experience entirely confirms his views, and I may most confidently say that I had arrived at and recorded similar opinions before I had become acquainted with those of M. J. Roux. At first sight it certainly seems a harsh proceeding to amputate at the hip for a compound fracture of the lower third of the femur, but if by doing so the risk of purulent infection be lessened, there can be little doubt but that it should be done. I am not prepared to say that it is absolutely necessary, at once, in all injuries; but in secondary cases where re-amputation is needed, from the bone in the stump having become affected, there can be no doubt that, as a general rule, it is entitled to serious consideration."

chiefly arises, in all probability, according to the views of M. Roux, the failures of secondary amputation for gunshot wounds of bones; and this also explains why, if after six months, or even a year, a surgical operation becomes indispensable, exarticulation, or removal of the whole of the diseased bone, is preferable to amputation in most, if not in all cases.

Acting on these conclusions M. Roux practised exarticulation in twenty-two successive cases, and with extraordinary success; for, as shown in the report, the patient in every instance recovered.[1] The exarticulation of the limbs were performed at the following joints:

Coxo-femoral . . .	4	times.
Scapulo-humeral . . .	13	„
Femoro-tibial . . .	1	„
Fibio-tarsal . . .	3	„
Metacarpo-phalangeal . .	1	„
Total .	22	„

The principal question involved in the views advanced by M. Roux is the following:—Is osteo-myelitis, or endosteitis, a necessary consequence of every gunshot wound of bones; and, on its occurrence, does the inflammation necessarily extend throughout the whole medullary system of the bone? For on the decision in respect to these questions must rest the practice or otherwise of sacrificing the whole bone by disarticulation when surgical interference of some kind is rendered obviously necessary from the protracted results of gunshot injuries. Now these questions were very fully discussed at several meetings of the Imperial Academy of Medicine at Paris in the year 1860, and were more particularly examined in a discourse delivered by Baron Larrey, which he

[1] Detailed reports of these cases will be found in a pamphlet, embodying the memoir on this subject read at the Imperial Academy of Medicine, published by Dr. Jules Roux, and entitled 'De l'ostéo-myélite et des amputations secondaires à la suite des coups de feu, d'après des observations recueilliés à l'hôpital de la marine de Saint-Maudrier sur les blessés de l'armée d'Italie.' Paris, 1860, pp. 82.

afterwards published.[1] Without entering into the course of his arguments I will quote the conclusions he arrived at; for they seem to be most consonant with facts, and with the opinions held by the majority of surgeons at the present time. Baron Larrey's conclusions are six in number,[2] and are as follows:

1st. Osteo-myelitis, after gunshot wounds, is more frequent than has been hitherto supposed, but is not inevitable, and in most instances is a means of cure.

2nd. It may either be limited to a given point of the bone, extend itself partially, or invade the whole of the bone more or less quickly.

3rd. Every rational mode of treatment must be adopted in the first instance. We are encouraged to do so because we know osteo-myelitis is susceptible of spontaneous cure.

4th. Sometimes it necessitates resection, and sometimes consecutive amputation; and sometimes, in certain cases, disarticulation is preferable.

5th. The existence of osteo-myelitis is sufficient to explain the want of success which occasionally follows partial operations in bones affected with this inflammation; but,

6thly. It does not justify the too exclusive proposition in surgery, that resections of joints, and amputations in the shafts of bones, are to be abandoned for disarticulation in all such cases.

When we remember the frequent instances which have occurred where balls have remained impacted in the spongy tissues of bones with impunity, without any evidence, when afterwards examined, of inflammation having been excited; when we remember the cases of gunshot fractures, even of the femur, which become consolidated under proper treatment, we cannot I think help concluding that osteo-myelitis after gunshot wound is not so universal in occurrence as has been argued by M. Roux, but must adopt generally the conclusions expressed by Baron Larrey on the subject.

[1] 'Des Amputations consécutives à l'ostéo-myélite, dans les fractures des membres par armes à feu.' Par M. H. Baron Larrey, Paris, Baillière et fils. 1860.

[2] Op. cit., p. 26.

I now wish to try and carry the settlement of the question of the treatment of chronic osteo-myelitis a step further in precision, especially in cases where want of success has seemingly followed partial operations from this cause. Many cases come before military surgeons in which, after amputation has been performed in continuity for gunshot injuries, or for the effects of osteo-myelitis consequent upon them, the portions of the limbs left subsequently present such diseased conditions as to necessitate further surgical interference in order to avert fatal consequences from the patients. In these cases, where the morbid state of all the tissues is manifestly due to the continued osteo-myelitic action subsequent to the amputation, what is the course to be pursued? The arguments of M. Jules Roux would urge most forcibly in these cases that exarticulation is the only treatment that can scientifically be adopted, and even according to the views propounded in the conclusions of Baron Larrey, these would appear to be the certain cases to which he refers in his fourth conclusion, where curative treatment in the first place, and consecutive amputation in the next place, having failed, disarticulation would be held to be the preferable course to follow. I have been led to adopt a different conclusion, but before giving my reasons I would first ask the attention of the Society to the following four preparations from the museum of the Army Medical Department.

Preparation No. 2918 (Plate II, fig. 1) shows the "upper portion of a humerus affected with necrosis after amputation at the middle of the arm in consequence of compound gunshot fracture. The patient, private J. Seaman, 1st Battalion Coldstream Guards, was wounded at Inkerman on November 5th, 1854. The primary amputation was performed in the Crimea. Secondary amputation at the shoulder was performed in England on July the 25th, 1855, by Dr. Bowen, Assistant-Surgeon, Coldstream Guards."

Osteophytic growth surrounds two thirds of the circumference of the sequestrum; it is limited to the parts exterior to the capsular ligament. The head and neck of the bone within the capsular ligament are in a state of osteo-porosis.

Preparation No. 2919 (Plate I, fig. 2) exhibits "Necrosis of the upper portion of the humerus of the right arm after amputation at the middle of the bone in consequence of compound gunshot fracture of the bones of the forearm."

"The patient, James Curds, 1st Battalion Coldstream Guards, was wounded at Inkerman, on the 5th of November, 1854. The first amputation was performed in the Crimea. The second amputation at the shoulder-joint was performed in England by Dr. Monro, Surgeon-Major, Coldstream Guards, on the 25th of July, 1855."

This specimen differs in some respects from the last. The sequestrum extends rather higher up, and terminates by a conical apex, the top of which rises to a level with the smaller tubercle of the bone. The osteophytic growth below is more extensive and extends rather higher. At the end of the stump it is thickly developed, leaving only a small central opening, through which a small portion only of the necrosed bone can be seen. Its condition indicates that a portion of the dead shaft had at some time in the course of treatment been removed from the end of the stump. The head of the bone is in a state of osteo-porosis.

Specimen No. 2916 (PlateII, fig. 1) is a section of the upper half of a humerus which was exarticulated at the shoulder, as a secondary operation to amputation in continuity, for a gunshot wound. The primary amputation was performed in May, 1855. In September, 1855, the patient, a young man aged 23 years, was admitted into Fort Pitt. The amputation wound was then cicatrized with the exception of two small openings. The patient's general health was indifferent. Subsequently to admission the whole stump was attacked by inflammation, followed by abscesses, which left two sinuous openings near the axilla, as well as general thickening of all the tissues of the stump. On November 11th a ring of dead bone, at the site of the amputation, was removed from the end of the humerus by incision. The patient's general health afterwards improved, but repeated inflammatory attacks occurred in 1856. In the beginning of April of that year an unfavorable action took place, and secondary ampu-

tation was decided upon as affording the only hope of saving life. This was performed on April the 19th. The patient died on May the 1st, 1856, of subacute peritonitis and other symptoms of pyæmia.

Preparation No. 3625 (Plate II, figs. 2 and 3) exhibits the head, neck, and part of the shaft of a right femur, illustrating the effects of endosteitis after a gunshot injury. Amputation was performed in the middle of the thigh soon after the injury.

The portion of the shaft exhibits a copious deposit of new bone, thickened towards the amputated end, and superficially presenting a strongly marked nodulated and laminated character. The osteophytic nodules and laminæ are of various sizes and forms of outline, but nearly all present a direction as if their growth had been from below upwards. In this outer covering of new bone there are four cloacal openings: three small and nearly circular in front, and one irregularly longitudinal, three inches in length, posteriorly.

The trochanters, neck, and head of the bone are free from the osteophytic deposit. They also appear free from taint of endosteitis, but from the absence of usual weight are doubtless to a certain extent affected with porosis.

Within the covering of new bone is a sequestrum of the original shaft, extending from the line of amputation up to an inch, or inch and a half, below the small trochanter.

History of the case taken from the museum records.

Private John Sole, 2nd Battalion Rifle Brigade, aged 24 years, was wounded on the 28th of Nov., 1857, at Cawnpore, by a grapeshot in the right leg. Amputation was performed at the middle of the thigh. (From the situation of the amputation it is to be presumed that the lower part of the femur, or knee-joint, were involved in the original injury, but no description of the wound received in India beyond the above is recorded.)

The patient was admitted into the detachment hospital at Gravesend on the 8th of July, 1858, having arrived with invalids from India by the ship "Argo." He was much emaciated. The stump had never healed, and was reported to have shown

signs of gangrene on board ship. There were several unhealthy looking sores on the face of the stump along the line of the amputation incision, and from them a profuse purulent discharge escaped.

The patient improved considerably for some time after his arrival at the hospital, gaining flesh and strength, and the sores assumed a more healthy appearance. Great pain was, however, occasionally complained of in the stump.

About the 20th of August, a little more than a month after admission, a swelling of considerable size appeared on the under surface of the stump, which, on being opened, gave issue to a large quantity of purulent discharge. Dead bone was felt through the opening thus formed.

He subsequently again lost flesh and strength, his appetite became bad, he suffered from bed-sores, the discharge from the stump was increased, and on the 11th of September he died.

At the post-mortem examination all the viscera of the chest and abdomen were found to be in a normal condition.

These four cases at first sight tend to support the doctrine laid down by M. Roux, as to the necessity of treatment by exarticulation. In all of them there has been a gunshot wound which has led to the necessity of amputation in the first place, but inflammation of the medullary membrane has advanced so that, ultimately, exarticulation has been resorted to in three of the cases, while in the fourth death occurred without operative interference. In none of them does the periosteal investment appear to have been involved in the inflammatory action beyond that degree of excitement which was serviceable for producing new deposit of bone. There is no reason to suppose that in any one of these cases the amputation was not performed sufficiently high to remove all the structures directly involved in the primary injury; nor is there any ground for supposing that the state of health of the patient, or any peculiarity in the operation itself, induced the subsequent unhealthy condition in the remaining portion of the shaft of the bone. We are left to conclude, then, that the endosteitis was the result of the shock of the original gunshot

injury, that it probably existed throughout the whole shaft of the bone at the time of the amputation, and did not subside afterwards in that portion which was left in the stump.

That endosteitis of such an extent may be set up by the *concussion* of a gunshot wound alone, irrespective of fracture or the effects of amputation, is illustrated by specimen No. $\frac{2}{8}\frac{0}{3}\frac{3}{7}\frac{4}{2}$. This preparation shows us that the entire shaft of the femur has been subjected to the action of endosteitis; yet the musket-ball, which led to this extensive mischief, simply penetrated the soft structures and struck the bone, without producing complete, or even a partial, fracture of its substance. (Plate I, fig. 4.)

But to revert to the practical question before named, viz., whether surgeons are justified by the circumstances of such cases as these in which endosteitis is presented in its most extensive forms, in concluding that exarticulation is the only means of cure.

I next ask your attention to the following cases, for they appear to me to be very instructive in reference to the question under consideration. They are, I believe, examples of cure by other means and with more satisfactory results, under circumstances precisely analogous to those just now referred to, in which the treatment of exarticulation was practised.

Case 1.—Sergeant H. Pearce, 103rd Regiment, invalided from India, was admitted into hospital at Fort Pitt, during the year 1862, on account of general endosteitis of the upper half of a femur after amputation in its middle third, which, though not the effect of gunshot, presented precisely the same features as if it had resulted from such a violent injury. The whole stump up to the hip presented a thoroughly diseased appearance; the soft parts were tumefied, œdematous, and painful, and were traversed by sinuses leading to dead bone in several directions. The patient's general health was very greatly impaired; he was in a very despondent state of mind; and, according to his own account, had suffered so long and so much that life was a burden to him. This case led to a

good deal of discussion as to its proper treatment. At one time, in consequence of the very unhealthy aspect of the whole stump, and partly influenced by the results of the experience of M. Roux in cases of a similar nature, at a general consultation of the staff of the hospital, exarticulation at the hip-joint was determined upon. Fortunately, however, before this serious operation was undertaken, a study of the museum preparations which have been brought before the notice of the Society, and some others of a similar kind, led me to determine to try as a preliminary measure a simpler operation, leaving the more severe one, if still required, as a last resource.

This consisted in opening the cicatrix of the amputation wound, and removing all pieces of necrosed bone that might be found within the remaining portion of the shaft. There would then be the opportunity of observing whether nature would not be equal to instituting a reparative action, and effecting the necessary contraction and consolidation of any new bone which had been thrown out around the diseased portion of the shaft, when these sources of irritation were removed. Complete success attended this effort. The dead portion of the shaft of the femur which reached up to the trochanters was extracted together with some small detached fragments, and the patient rapidly improved in all respects afterwards. Eventually an artificial limb was applied to the stump and worn without inconvenience, and the patient left the hospital walking with the aid of a stick.

The principal portion of the bone removed is shown in Plate II, fig. 4.

Case. 2.—A soldier was under the care of Assistant-Surgeon Dr. Becker, then acting as pathologist in the hospital at Scutari, during the Crimean war, in which a sequestrum, six inches in length, formed by nearly the entire circumference of the shaft of the femur, was extracted. The end of the sequestrum had previously protruded from the face of the stump. This morbid condition followed amputation after a gunshot wound, and continued osteo-myelitis in the remain-

ing portion of the bone. The removal of the dead bone was followed by a sound state of the stump in about three weeks.

Case 3.—Corporal John Roberts, R.A., æt. 17 years, had his left leg completely smashed by a round-shot just above the knee, at Alumbagh, on the relief of Lucknow, on the 25th of September, 1857. The limb was amputated four inches and a half below the trochanter major, a few hours after the injury, by Dr. F. Innes, Surgeon of the 84th Regiment. A fortnight after the operation the healing process was sufficiently advanced to allow of the patient's removal in a dhooley to Cawnpore. He was under hospital treatment for a fortnight at Cawnpore, and was then sent on to Allahabad. He continued improving, and six weeks after his arrival at this last-named station the amputation incision of the stump closed completely. The cicatrix was not, however, firm on pressure, and a short time after having thus apparently become healed, a small opening appeared in it, through which there gradually became protruded about an inch of bare bone, comprising the whole circumference of the femur. This was moved backwards and forwards every morning, the movement causing considerable pain towards the hip-joint, and within a fortnight from its first appearance the sequestrum was sufficiently loosened to admit of extraction. It was three inches and a half in length; smooth at the amputated end for about an inch, but roughened on the surface for the remaining two and a half inches; and terminated in three sharply pointed spiculæ. The stump then became firmly cicatrized.

A careful examination of this patient was made in October, 1862, five years after the amputation. He was at this date employed in the arsenal at Woolwich, his work lasting ten hours a day, and his duty being to stoop and put in their places fusees after having carried them some distance. The following is the description of the condition of the stump at this time:—"The present bony formation of the stump is of the same length from the trochanter major as the original upper half of the amputated femur. The extremity of the

new bone appears hard and firm, but is at least double the size in all directions of the original bone. The integument of the stump is quite sound, and there is no pain felt in it except on change of weather; but occasionally the new skin feels tight as if the new bone were too large for it. The flap appears well developed although the muscular portion of it does not entirely cover the bone. The amount of strength and endurance of pressure in the stump is very considerable; with a light artificial leg he has walked fourteen miles without difficulty. He generally, however, wears a heavy wooden leg while at his work. There is perfect motion of the hip-joint.

The result of the cases I have just named where stumps have been preserved without exarticulation or further amputation; and a careful examination of the diseased conditions in the preparations I have brought before the Society, where stumps have been removed by exarticulation, or where death has occurred, as in the case from which the preparation of the femur was taken, without operative interference; have led me to the conclusion that in all cases, however severe, of endosteitis and endosteitic necrosis after gunshot injuries, before resorting to the extreme measure of exarticulation as urged for all cases by M. Jules Roux, and recommended in certain cases by Baron Larrey, the opportunity of effecting a cure by careful and complete removal of sequestra should invariably be tried. The judgment of the surgeon will be tested in determining the precise period when the process of sequestration is sufficiently advanced to admit of operative interference with advantage; and his qualities as a physician will be put to the test in the support he must afford to the patient's constitution while subjected to the irritation inseparable from this protracted ordeal. But when the period has arrived of nearly complete sequestration of the dead bone, then the stump should be opened if amputation has been previously performed; or the steps taken such as are employed in ordinary necrosis if no amputation has been performed; and the necrosed portions be entirely removed, and the case treated on general principles. I now feel certain that if this treatment had been practised instead of exarticula-

tion in the following case represented by preparation No. 2418 (Plate I, fig. 3), on which I myself operated, the stump might have been saved and a cure effected without exarticulation. On examination it will be found that this preparation exhibits a condition which exactly corresponds with the condition of the preceding specimens, though it did not result from an injury by gunshot. The patient, Private Noah Gowan, of the 77th Regiment, injured his wrist in India by a fall, on the 25th of December, 1860. Amputation of the forearm had to be performed on the 9th of January, 1861. Unhealthy action followed, accumulations of pus took place in the arm and elbow-joint, and amputation was again performed in India about three weeks afterwards in the middle of the upper arm. He arrived from India at Fort Pitt on the 25th of April, 1862. The remaining portion of the humerus being then in the necrosed state exhibited in the preparation was removed by exarticulation on the 16th of May following. He was discharged with the parts soundly cicatrized on the 23rd of July.

I have no doubt also that a similar mode of proceeding might have saved the stumps to which the other preparations brought before the Society belonged. In the cases represented by preparation No. 3625 (Plate II, figs. 2 and 3), and by specimen No. $\frac{2}{4}\frac{0}{3}\frac{3}{7}\frac{4}{3}$ (Plate I, figs. 4 and 5), removed post-mortem, not only the limbs but perhaps the lives of the patients might have been preserved by the removal of the bone sequestra, and the arrest and cessation of the irritation caused by their lodgement. The removal of sequestra under such circumstances presents no greater difficulties than they do in cases of necrosis from ordinary causes; in the instances of stumps, their removal, indeed, must ordinarily be an exceedingly easy operation.

It is not a matter of such great importance to avoid exarticulation of a humeral stump as it is of a femoral stump; for the one is almost without danger to life, while in the other life must always be seriously endangered. But even a humeral stump, if the power of the pectoral, latissimus dorsi, and other muscles employed in approximating it to the side,

has not been lost, is often of great value to the possessor. And independent of the dangers of a hip-joint amputation, though these are lessened when practised as a secondary operation, the important use of a thigh-stump for adapting and securing the mechanical contrivances employed to sustain the weight of the body, and to assist in progression, cannot be over-estimated.[1] Any surgical means, therefore, by which such portions of the extremities can be saved are well worthy of study on the part of the surgeon, and as far as the patients themselves are concerned, any ordinary expenditure of time and endurance will be well repaid if a successful issue be obtained eventually.

There is one condition often allied with these cases of protracted endosteitis which might make a surgeon hesitate to trust to such an operation as I have described for his patient's safety and cure. If the articular head of the bone in each of the cases I have brought to the notice of the Society be examined, it will be found that it yields more readily than natural to the pressure of the finger. The cartilaginous surface seems to have lost its own strength and to have lost support beneath; it can readily be bent inwards. The weight of the bone is considerably diminished, and on section it is found that there really is a very considerable absorption of the earthy portion of the structure, and that it has an abnormally cellular appearance; in short, that it is in that condition to which the name of *Osteo-porosis* has been given.

I believe, however, that this condition will not interfere with a successful result in those cases where the necrosed sequestra have been completely removed. We have not the

[1] In the 'Edinburgh Medical Journal' of this month, November, 1864, is reported a successful case of exarticulation at the hip-joint, however, in which the patient, a native of India, does not appear to suffer much from the loss of a thigh-stump. "He uses crutches and gets over the ground rapidly, and goes to work regularly as a tailor." The exarticulation was performed by Professor Fayrer, of the Medical College Hospital, at Calcutta, for osteo-myelitis, following amputation of the thigh at the lower third, rendered necessary by injury to the knee-joint from the kick of a horse.

means of affording demonstrative proof of the existence of the condition above stated in those cases in which success follows the modified operation. I have not the least doubt that the head and neck of the femur, in the instance of Sergeant Pearce, from having been so long unused, and from having been subjected to the irritating effects of the endosteitic inflammation in the neighbouring shaft for nineteen months after the operation of amputation, were in a state of osteo-porosis at the time the sequestra were removed; but that this condition was subsequently repaired when the sources of irritation were taken away. And in proportion as such a stump is employed in its movements with an artificial limb, and thus becomes subjected to the stimulus of use, there can be no doubt that a more and more healthy condition of the bone is gradually obtained.

The year before last I removed a foot in a scrofulous boy, for extensive caries of the ankle-joint, and, on sawing off the two malleoli, I observed that the extremities of both the tibia and fibula were extensively affected with fatty osteo-porosis. The bone was quite soft, honeycombed, and oily fat-cells were visibly occupying the hollow spaces in the porous extremities; yet this stump healed, and subsequently bore the weight of the body and admitted of progression without inconvenience. Here, doubtless, the cancellated tissue gradually recovered a healthy condition and the ends of the bones became firm, by the deposit of new bone, under an improved condition of general health, and the removal of the source of irritation which had previously existed in their immediate neighbourhood.

In conclusion, I would observe that while adopting generally the views of Baron Larrey in reference to the nature, progress, and treatment of osteo-myelitis after gunshot injuries before quoted, the following appear to me to be fair deductions from the facts and observations which have been noticed in this paper.

1st.—In gunshot injuries of bone it will be found for the most part what might be anticipated from the intimate con-

nection which exists between the periosteal and endosteal investments of the bony tissues, and from the violent general mischief effected by the stroke or passage through them of a projectile—that all the structures participate not only in the immediate local destruction, but also in the extended inflammation which follows, whether the inflammation after a time subsides and terminates in repair, or whether it continues in a chronic form.

2nd.—There exists this difference between the inflammation of the endosteum and that of the periosteum; that of the endosteum has a special tendency after gunshot injuries to degenerate into a chronic condition analogous to that of suppuration in other tissues to extend itself along the cancellated structure, and thus to produce disintegration and death of the bony substance; that of the periosteum, at the same time, will exist only to such a degree as to cause it to exert a protective influence by the formation of new bone around the diseased tissues, just as in ordinary cases of necrosis from other causes.

3rd.—If amputation in continuity be performed while the endosteum is suffering from the inflammatory irritation excited by the violent injury to which the whole bone has been subjected, especially when this has assumed a chronic form, the endosteitis will most probably still pursue its course, even though the divided soft parts may at first become healed, slowly inducing death, more or less extensive, of bony tissue, and in time the usual consequences of such a condition throughout the whole stump.

4th.—The morbid condition of the endosteum does not usually extend from the shafts of bones into their apophyses.

5th.—When amputation has been followed by these consequences, exarticulation should not in any case be resorted to for the removal of the diseased stump, until the effect of complete removal of every particle of the dead bone by proper surgical measures has been ascertained.

6th.—Experience shows that although a patient's constitution may be greatly impaired by the prolonged local diseased action to which it has been subjected, and though

there may be every reason to conclude that the articular extremity of the bone is in the condition understood by the term "osteo-porosis," yet the complete removal of the endosteitic sequestra may speedily be followed by restoration of the general health, and by a condition of the stump so sound and firm that it may be applied to any purpose of utility for which it may be competent, according to its length and position.

DESCRIPTION OF PLATE I.

FIG. 1.—Upper portion of humerus affected with endosteitic necrosis. Head and neck of bone in a state of osteo-porosis (the principal sequestrum is represented rather lower than natural).

FIG. 2.—Ditto, ditto. (The outer casing of the new bone has been accidentally broken across at *a*.)

FIG. 3.—Upper portion of a humerus exarticulated for endosteitic necrosis, after amputation for injury by a fall, in India. The necrosis does not extend into the tuberosities, neck, or head of the bone. These parts are, however, in a state of osteo-porosis.

FIG. 4.—A femur with its entire shaft in a state of endosteitic necrosis, consequent on concussion from the stroke of a musket-ball; anterior view. No fracture produced by the projectile. The sequestra, which are visible through the cloacæ, are limited by the apophyses of the bone; *x* and *y* show the limits of the necrosed bone within the shaft.

FIG. 5.—Ditto, ditto. Posterior view. The sequestrum partially visible at *a* does not extend into either trochanter or into the neck of the bone.

Fig. 1.
(N° 2918.)

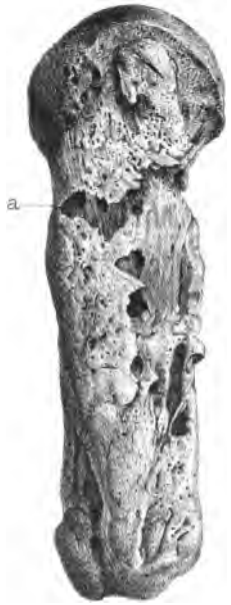


Fig. 2.

Fig. 3.

Fig. 4.

Fig. 5.

C. H. Ford.

W. West imp

DESCRIPTION OF PLATE II.

FIG. 1.—Section of the upper half of a humerus, exarticulated at the shoulder for the effects of endosteitis after amputation in the middle third, for a gunshot wound. *a* points to the unremoved portion of sequestrum; *b* points to a small sinus; *c* points to the articulating head and neck of bone unpenetrated by the necrosing action. Near *a* on an aspect not shown in the plate was a large cloaca and sinus.

FIG. 2.—Head, neck, and part of the shaft of a femur, illustrating the effects of endosteitis after amputation for gunshot injury: anterior view. The necrosed sequestra partially seen through cloacæ at *a* and *b* do not extend higher than the shaft.

FIG. 3.—Ditto, ditto. Posterior view. The sequestrum partially seen at *a* ceased just below the smaller trochanter. The head, neck, and trochanters are lighter than normal, probably in a state of osteo-porosis.

FIG. 4.—Sequestrum removed from the thigh-stump of Sergeant Pearce, July 3rd, 1862 (see abstract of case, p. 54).

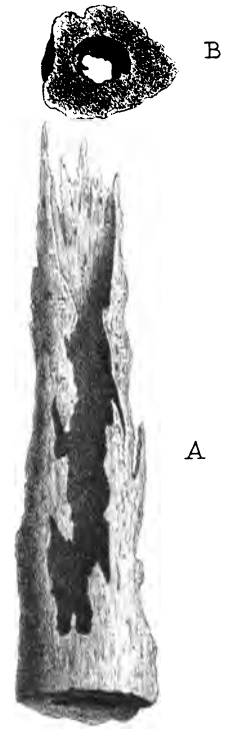
A. Side view (length of natural size, five inches).
B. End view.

G.H. Ford.

Fig. 1.
(N° 2916)

Fig. 2.

Fig. 3.



W. West imp.

Fig. 4.

CASE

OF

ANEURISM BY ANASTOMOSIS OF THE SCALP,

TREATED SUCCESSFULLY BY LIGATURE OF THE COMMON CAROTID AND SETONS.

BY

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Received Jan. 19th,—Read March 14th, 1865.

The difficulty of treating aneurism by anastomosis, where it extends over a large portion of the scalp, is my reason for bringing before the Fellows of this Society a case of perfect cure by ligature of the common carotid and the introduction of setons.

The patient, a married female, æt. 28, had been suffering from dilatation of the vessels on the right side of the scalp for upwards of eight years. The disease was at first of limited extent, implicating only the vessels over the upper portion of the parietal bone, but it gradually spread towards the temporal fossa, behind the ear, and in the direction of the occiput. The vessels continued to enlarge, and those over the frontal bone and in the palpebræ of the right eye also became slightly affected.

A fortnight ago, a small ulcer formed over the parietal protuberance, and hæmorrhage occurred from it on several occasions, which was at first easily arrested by lint and bandage. The last bleeding, however, having been more severe than its predecessors, she sought and obtained admission into the Manchester Royal Infirmary.

She came under my notice for the first time on the 19th of May. Her countenance was pale, and plainly indicated that she was just recovering from hæmorrhage which had taken place a few days before. On the removal of the bandage I found the disease to extend over all the right side of the scalp. The temporal artery and its branches, with the exception of those distributed around the eye and the forehead, which, though visibly distended, did not pulsate, were much enlarged, some of them to the size of the little finger, and communicated to the hand a distinct arterial thrill. The occipital artery and its branches were also similarly affected, though in a less degree. The pulsation was only slightly checked by pressure on the trunks of these vessels, but was completely suspended by compression of the common carotid. I attempted to remove the lint covering the ulcerated surface, but arterial hæmorrhage supervened to so great an extent that I immediately replaced the bandages, and invited a consultation of my colleagues.

On the following day, having taken the precaution to have the requisite appliances for the arrest of hæmorrhage at hand, I removed the compress in the presence of Dr. Eason Wilkinson, Mr. Dumville, and several of the resident members of the hospital staff. Profuse bleeding followed from the ulcerated surface, which occupied a space at the upper part of the parietal bone about an inch and a half in diameter. Pressure with the fingers failed to stop the effusion of blood, escaping, as it did, from countless sponge-like orifices. Lint steeped in a solution of perchloride of iron was also applied and the carotid compressed, but the blood continuing to flow, with the consent of my colleagues, I proceeded to place a ligature on the trunk of the common carotid, above the omo-hyoid muscle, which had the desired effect. The patient was

seized with a convulsion at the commencement; some delay, in consequence, occurred in securing the vessel, and it was feared she would scarcely survive the completion of the operation. In consequence of the urgent nature of the case chloroform was not given. Essence of beef, yolk of eggs, and brandy were afterwards freely administered, and the head kept very low.

May 22nd.—The condition of the patient was favorable, and she had partaken freely of nourishment. Pulse 100; bandages and compress removed from the head; the ulcerated surface dry, with the exception of a few drops of arterial blood exuding from its edges where the lint had adhered. No pulsation perceptible in the vessels of the temple or any part of the scalp. Wound in neck healthy, suppurating. Simple dressing and a light bandage applied to the ulcer.

26th.—Progress satisfactory. Very feeble pulsation can be felt at the temple, near the ear; the vessels of the scalp soft, flaccid, and apparently bloodless.

28th.—On removing the dressing, several drops of arterial blood flowed from the edge of the ulcer. Skin over the eyebrow disposed to slough from pressure of the bandage.

Four setons of worsted about four inches in length were passed through the diseased structure, one across the temporal fossa, the others through the parts of the scalp where the vessels were most distinct.

June 3rd.—Still progressing favorably; no increase of pulsation; setons suppurating. The ligature around the carotid came away to-day.

7th.—Two setons removed.

9th.—Some vessels near the original sore distended and slight pulsation in them discernible. Seem to be supplied with blood from the opposite side of the scalp. Three setons inserted into the largest vessels. An hour afterwards the pulsation had ceased and the blood in the vessels appeared to be coagulated.

18th.—All the setons freely suppurating. The vessels into which they pass enlarged, and on pressure a free discharge of pus. Two setons withdrawn, arterial hæmorrhage fol-

lowing, but easily checked by a light compress of lint. No pulsation felt in any part of the scalp. The slough over the eyebrow separated, and the sore looking healthy; general health good.

19th.—The remaining setons removed; free discharge of pus, but no further hæmorrhage.

27th.—The wound healing. All parts of the scalp firm and no vessels enlarged, except at the posterior part of the parietal bone, where one, about an inch and a half in length, can be distinctly traced pulsating slightly. A seton inserted along its entire length.

July 13th.—The seton removed and a quantity of pus escaped.

August 6th.—No return of pulsation since the introduction of last seton. The original sore quite healed, that on the forehead contracting.

10th.—Part of the scalp behind the ear sloughing; has been slightly feverish the last few days.

15th.—The slough has separated, and having extended to the bone, a portion of it, about one third of an inch in diameter, is denuded. In other respects quite well. Left the hospital to-day.

December 27th.—Scalp carefully examined, but not the slightest trace of the disease could be discovered. The exfoliated bone almost separated.

Remarks.—The success of the plan of treatment adopted in the above case affords a prospect of bringing this hitherto unmanageable complaint more effectually under the control of the surgeon.

At the time of the patient's admission into the hospital the disease had arrived at a stage when prompt measures were absolutely necessary for the preservation of life. On a careful examination I was satisfied that the circulation through the vessels could only be completely stopped by ligature of the common carotid, and, therefore, as soon as hæmorrhage supervened, finding the usual remedies inadequate, I immediately resorted to that operation.

But the unfavorable results which have frequently followed

deligation of the carotid for aneurism by anastomosis of the scalp induced me not to rely solely on this method of treatment; and the further progress of the case showed that if other means in addition had not been employed, no permanent benefit would have resulted from the operation. Indeed, the repeated failures of ligature of the carotid, as well as of other arteries supplying this variety of aneurism in other parts of the body with blood, prove to demonstration that it can no longer be regarded as a curative measure except in very mild forms of the disease.

This need excite no surprise, for the operation, to be successful, must either permanently arrest the circulation through the diseased vessels, or lead to their obliteration, conditions which, however probable where the disease is confined to a single vessel and assumes the ordinary form of aneurism, are not likely to follow when several are affected, as in the present case, involving the entire temporal system with its arteries, veins, and capillaries.

For the blood in the vessels after deligation does not coagulate, but readily finds its way into the general circulation, and the vessels remaining unchanged become again distended as soon as the circulation through the anastomosing branches is re-established.

But, despite these drawbacks, deligation even when not required for the suppression of hæmorrhage has its advantages, the temporary interruption which it causes to the circulation through the diseased structures, affording a favorable opportunity for the application of other remedies. I resorted, therefore, to the use of setons as soon as I was satisfied by the usual indications that the scalp was supplied with blood sufficient for reparative purposes.

The setons were first passed across the trunk and branches of the temporal and occipital arteries, also through the parts of the scalp where the vessels appeared to be most dilated. Others were afterwards introduced wherever any return of pulsation showed itself. Some of the later ones were inserted in vessels dilated to the size of the little finger, and in which a slight arterial thrill could be perceived. The earlier

setons seemed merely to excite adhesive and suppurative inflammation in those structures which they penetrated: but where the vessels had become distended with blood, the inflammatory action was preceded by coagulation, which took place shortly after the introduction of the setons.

Fine needles, in length about four inches, threaded with worsted of sufficient bulk to prevent the escape of blood, were passed freely into the vessels, precaution being taken both in their entrance and exit by the penetration of the skin at a short distance from the vessels to prevent the entrance of air. The extent of structure traversed by the setons would be about thirty-six inches.

It is unnecessary to dwell on the futility of trusting to setons only in the treatment of these cases. They have so repeatedly failed that their success in the present instance must be attributed to the quiescent state of the circulation produced by the ligature of the carotid. In confirmation of this view, I may briefly refer to the case of a young lady who was under my care several years ago, whose index finger and thumb had become, through enlargement of the vessels, twice their natural size. The disease commenced at the extremities of the fingers, and gradually extended itself towards the hand. Some of the vessels were in circumference as large as goose-quills, and gave a livid bluish appearance to the fingers. Not the least pulsation or arterial thrill could, however, be discovered, and the vessels were partially emptied of their blood by pressure. Three fine worsted setons were passed from the hand to the apex of the finger, and remained there for three weeks. Others were inserted at intervals. At the end of six months all evidence of the disease had disappeared. Slight traces of the affection presented themselves about eighteen months afterwards, which two small setons completely removed.

Deligation of the arteries, therefore, prior to the insertion of setons, does not appear necessary in all cases of aneurism by anastomosis. The disease being an affection of the arteries, veins, and capillaries, varying in its character according to the extent to which each of these structures is implicated, deligation seems to be required where the arterial tissue is

principally involved, or where enlargement of the capillaries and minute arteries has taken place to such a degree as to enable the force of the heart's action to communicate its impulse through the capillaries to the blood circulating in the veins.

Similar considerations will also determine the extent of deligation, which, except where severe hæmorrhage occurs, need in the majority of cases only be applied to the smaller arteries.

Though setons were employed in the case now related, galvanism, the injection of perchloride of iron, and other similar agents, may, in some instances perhaps, be advantageously substituted; and even the risks attending ligatnre of the arteries may by instrumental or digital compression be occasionally obviated.

CONGENITAL HYDRONEPHROSIS

IN A BOY FOUR YEARS OLD;

REPEATEDLY TAPPED. RECOVERY.

BY

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Received Dec. 23rd, 1864.—Read March 14th, 1865.

J. R— first came under my notice in March, 1863. He was then three years and four months old.

His mother stated that his stomach had been large ever since birth; when three months old a doctor who saw him recommended tapping, but the parents would not allow it.

When sixteen months old he was a patient in the Leeds Infirmary, and the doctor there thought it should be tapped. The operation was decided on, but in three days the swelling had subsided so much that the operation was not performed.

For twelve months he had been much in the condition in which I first saw him, the abdomen, however, being sometimes larger than at others; at different times the circumference had varied from twenty-four to twenty-seven inches. The child had always had a large appetite; nothing abnormal had been observed in the alvine or urinary secre-

tions. His father's family was consumptive, and he had several brothers and sisters, all of whom were said to be delicate.

On April 2nd the following note was made of his condition.

Patient is small for his age; he has a rickety head and limbs. He sits up in bed, seems comfortable, and has no dyspnœa. His limbs are moderately nourished. Pulse 104, tolerably full and regular; respirations thirty in the minute.

The heart's apex is raised, beating almost close to the nipple; its sounds are normal; lateral diameter of thorax narrow from rickets.

Abdomen very large and tense; skin smooth and shining; superficial veins extremely large and distinct. There is lateral bulging, more marked on the right than on the left side. The ribs are pushed up, and the ensiform cartilage is tilted upwards; the greatest circumference of the abdomen is at a point midway between the ensiform cartilage and the umbilicus, where it measures twenty-six inches. From the tip of the ensiform cartilage to the umbilicus is seven inches and a half. The swollen abdomen fluctuates very distinctly on palpation. Between the ensiform cartilage and the umbilicus there is a tympanitic percussion-note; below the umbilicus the percussion-note is duller, and in the right flank there is absolute dulness.

Over the abdomen generally there is an irregular doughy sensation on manipulation, but in the right hypochondrium there is a sense of firm resistance. The liver and spleen cannot be detected, and no tumour can be made out. There is no sign of dropsy except in the abdomen; urine is acid, free from albumen, having a specific gravity of 1016, and is passed in fair amount.

The case at this time was not understood, and it was supposed there was fluid in the peritoneal sac, but from what cause could not be conjectured. There was no reason to suspect hepatic or mesenteric disease.

A more careful consideration of the physical signs would have shown that the fluid accumulation was not in the

peritoneal cavity. The extreme dulness of the percussion-note, the firm resistance offered on pressure in the right flank, and the want of symmetry in the swelling, were not compatible with simple ascites.

Under the false impression just referred to the patient was treated with diuretics and purgatives. But no benefit resulted. The abdomen increased in size until it measured twenty-eight inches and a half in circumference, and it was noted that the right half of the abdomen measured an inch and a half more than the left. This was on the 16th of April. His abdomen was so heavy that when he was lying down he could not raise himself up again.

On the 17th of May the abdomen had diminished in size, and measured about twenty-four inches. As his health was not suffering, and his respiration not much interfered with, he was allowed to go home.

On the 8th February, 1864, he again came under my care at the Hospital for Sick Children, and on the 17th the following note was made of his condition.

His general health is tolerably good, though his limbs are weak, and from the weight of his abdomen he is unable to walk.

The abdomen is almost globular, but the swelling is greater below than above the navel, and greater on the right side than on the left. The circumference midway between the navel and tip of the xiphoid cartilage is twenty-seven inches. The umbilicus is not protruded, an enlarged vein runs from the right side of epigastrium towards the navel. Midway between the navel and the end of the ensiform cartilage there is a distinct sulcus (more marked on the left than on the right side) moving with respiration. This sulcus is found on close examination to correspond to the lower portion of the transverse colon, which is movable, and can be pushed down to within an inch and a half of the navel; it lies on a firm globular swelling, which is elastic and fluctuating. Something like the cæcum can be felt in the right iliac fossa in front of the same mass. The surface of the swelling is everywhere smooth and even, and the percussion-note is absolutely dull. There

is perfect fluctuation throughout every part of it. The prominence of the tumour can be most distinctly seen and felt in the right lumbar region; there is nothing corresponding to it on the left side. Distinct fluctuation is transmitted from the navel to the right lumbar region. From the notch of the sternum to the end of the xiphoid cartilage measures three inches and five-eighths; from the xiphoid to the navel seven inches and three-eighths, and from the navel to the pubes measures three inches and five-eighths. The upper limit of hepatic dulness is one finger's breadth below right nipple. The heart's impulse is felt just above and inside the left nipple. The urine is clear, pale, and free from albumen.

It was now obvious that the boy had a large cyst, with not very thick walls, springing from the right side of his abdomen, very likely connected with the right kidney, and that the large intestines were in front of it.

It was resolved to tap the cyst to relieve the patient of his burden, which prevented his walking and interfered with his respiration.

A trocar was introduced half an inch below and an inch and a half to the right of the navel. There escaped 102 fluid ounces of clear yellowish fluid, faintly acid in reaction, having a specific gravity 1008. It contained no albumen and there was no sediment. It had all the characters of dilute urine.

On chemical analysis it was found to contain 20·304 grammes or nearly 313 grains of urea; 5·9 grammes or 81 grains of chloride of sodium, and 0·457 gramme or 7 grains of uric acid. It also contained phosphates and sulphates.

From this analysis it was concluded that the cyst was the dilated pelvis of the right kidney, due probably to malformation or obliteration of the ureter on that side.

After the operation he was left undisturbed, and there were no bad symptoms consequent upon the tapping. For twenty-four hours after it he had retention of urine, and the bladder was relieved by the catheter.

Four days after the operation, the abdomen measured twenty-three inches and a half; his tongue was clean and moist, and his skin cool; pulse 118.

On 25th of February, a week after the operation, the abdomen was much flatter than it had been; it measured from the xiphoid cartilage to the navel only five inches and a half instead of seven and three-eighths, and in its greatest circumference twenty-three inches and seven-eighths.

On palpation of the abdomen, the condition of the parts was much less obvious than before. Still, a line was made out passing from right to left, and from above downwards, separating a less resistant upper part from a more resistant lower one; the latter was dull on percussion, while the former was tympanitic; owing to the flaccid state of the abdominal walls, fluctuation could not be well made out. Fæcal masses could be felt in the ascending, transverse, and descending colon. The heart's apex was felt a little nearer the middle line, but not lower than before.

The swelling soon increased, and by the 5th of March the abdomen measured twenty-five inches and three-quarters.

On the 15th of March an experiment was instituted to determine whether the fluid which was re-collecting in the cyst was of the same character as the urine. As the abdomen now measured twenty-nine inches, it was thought desirable again to tap him; but before doing so he was to take for two days ten grains of the ferrocyanide of potassium twice a day.

On the 16th the urine gave a very dark colour on the addition of the sesquichloride of iron, owing to the ferrocyanide passing off by the kidney.

On the 18th a small quantity of fluid was let out of the cyst, in order to compare it with what was drawn off by the first operation, and to see if it contained ferrocyanide of potassium.

It was now found to be neutral in reaction, slightly turbid from pus, and it contained a trace of albumen. It exhibited no reaction with the sesquisalts of iron. It contained proportionately only half as much urea as the first specimen.

The circumstance that the fluid drawn off contained no ferrocyanide, although the urine did, and that the urea had diminished in amount, made it doubtful whether the fluid which was re-formed had been secreted by the kidney or was from the wall of the cyst alone; on the latter supposition the urea which it contained must have come from the fluid left after the first operation.

By the 29th of March the abdomen had attained a size as large as ever it had; it measured twenty-nine inches and three-quarters. After a consultation it was thought advisable again to tap the cyst, and establish a permanent fistula. At this time the boy had frequent attacks of dyspnœa at night, which obliged him to sit up in bed.

On the 7th of April a small piece of caustic potash was applied to the abdominal wall in the situation of the former punctures, with the view of setting up adhesions between the cyst and the parietes, before opening the cyst.

During the two following days the boy became so weak and ill that it was not considered safe to wait for the gradual action of the caustic; paracentesis was accordingly performed with a trocar, and after drawing off the contents, the canula was left in the sac, and its free extremity was corked up. About seventy-four ounces of fluid were drawn off in this operation. It was now of a deep chocolate colour, nearly opaque, alkaline, and depositing about one quarter of albumen on boiling and the addition of nitric acid.

Under the microscope numerous blood-discs and pyoid cells were seen. It contained relatively more urea than the first specimen, namely, 6·5 grammes in 1000 cubic centimeters of fluid instead of 6·2 grammes in the same quantity.

For five days—from the 13th to the 17th March—the urine was carefully saved, and found to exhibit the following characters:—it was free from albumen, sometimes faintly acid and sometimes neutral in reaction. The average quantity passed in twenty-four hours during the five days was only 306 cubic centimeters, or between ten and eleven ounces. Its specific gravity varied from 1019 to 1026. He passed on the

average 7·8 grammes, or 120 grains of urea per diem, and of chloride of sodium 1·524 gramme, or 23·4 grains.

Considering his weight (twenty-nine pounds) and age, there was a decided deficiency of water and solids in the urine; there was not more than half the normal amount of water, about two thirds of the normal quantity of urea, and one quarter of the normal amount of chlorine; estimated on the scale given by Dr. Parkes in his treatise on the urine.

The fluid drawn off by tapping contained urea in the proportion of rather less than a third of what his urine passed by the bladder contained.

On the 10th April (the day after the third tapping) he was very ill. His pulse was 190; he complained of much headache and abdominal pain. His temperature was 104·75° Fahr.; tongue furred, white, and dry.

The canula was withdrawn, and a piece of gum elastic catheter substituted for it. Fifty-two ounces of fluid were drawn off, of a specific gravity of 1008, and with other characters exactly similar to what had been obtained on the previous day. Almost directly after this he passed by the urethra about eight ounces of fluid, specific gravity 1015, of a colour midway between that of the fluid drawn from the cyst and that of his urine. In half an hour later he passed five ounces of fluid almost identical in appearance with that of the cyst. It had a specific gravity 1012; contained an albuminous precipitate, when boiled and acidulated, filling nearly one quarter of the test-tube, and it had an alkaline reaction.

The previous day his urine had been clear, faintly acid, without albumen, and had a specific gravity 1017.

This was the first occasion on which we had any distinct evidence that the cyst and the bladder ever communicated, although the mother's statement that, on several occasions after extreme distension of the abdomen, the swelling had suddenly subsided, made it probable that such a communication had existed.

On the 11th April there was a marked increase in the quantity of urine passed, and it was of a pale-brown colour

and faintly turbid; had a specific gravity 1014, and contained a trace of albumen. It was quite clear from these marked changes in the urine that a free communication had been suddenly opened up between the bladder and the cyst.

On the 13th April he was better, had no pain; pulse 156; temperature 99°. A good deal of fluid dribbled from the wound. Urine very slightly turbid, was faintly acid, and contained a mere trace of albumen.

On 15th April, pulse 130; much emaciated. There was much fluid, sometimes thin sometimes thick, dribbling from the canula; the quantity was estimated at two quarts in twenty-four hours. The abdomen was not tender, and measured twenty-one inches and seven-eighths. He did not pass any urine for thirty-six hours. The cyst was now injected with warm water and a weak solution of Condy's fluid, to correct fœtor and cleanse it.

On 21st April the boy's general condition was much the same. All the fluid that escaped from the cyst in twenty-four hours was saved, and measured 345 cubic centimeters or about 12 ounces; it was light-coloured, turbid, very fœtid, highly albuminous, and contained about thirty-six grains of urea. A large quantity of pus was also present in it.

On the next day the discharge from the cyst entirely ceased, and on introducing a probe to the wound it was obstructed at a distance of about an inch and three-quarters from the entrance. From this time the swelling again rapidly increased in size until, on the 2nd May, the abdomen measured twenty-seven inches and one-eighth. A tumour was now noticed in the *left* flank, just outside the quadratus lumborum, reaching from the twelth rib to the crest of the ilium, about two inches across. It was dull on percussion, did not fluctuate; the surface and edges felt even. This was no doubt the left kidney.

On the 7th May, the tension being very great, the abdominal wall gave way at the seat of the old punctures, and a quantity of fluid escaped. A long curved canula was now placed in the cyst with the view of keeping it permanently open. It was found impossible to do this; the

fluid soon ceased to flow through the canula, and the wound healed.

On the 30th May it was determined to establish an opening by means of caustic potash in the right loin.

On the 6th June the abdomen measured twenty-nine inches and a half; the caustic had formed a deep slough, but had not opened into the cyst. A trocar was introduced through the eschar, and about two quarts of thin, fetid, purulent fluid escaped.

It was found impossible to keep this wound open; it healed up, and the boy's condition became much the same as before.

On 27th June he was regaining flesh and strength.

A little later he had an attack of measles, which he passed through without a drawback.

On 24th September it was noted that he had much improved in his general health; was quite fat, and could walk with slight support.

The abdomen measured twenty-five inches and three quarters in circumference, and from the ensiform cartilage to the navel five inches and a quarter. It was much softer and more yielding than it had been; it swayed over to the flanks to a certain extent, and was very flat on the top. The swelling in the left loin was not now to be felt distinctly.

The urine was saved for twenty-four hours, and found to have a specific gravity 1013, and to measure about seventeen fluid ounces. It was pale, turbid, and very fetid. The microscope showed—(1) prisms of triple phosphates; (2) vibriones; (3) pus and pyoid cells, and small casts containing pyoid cells, very fatty. He was now sent to the convalescent establishment at Mitcham.

Since that time he has continued to improve; can walk about well, and has gained flesh. The abdomen varies in size from time to time, but has never been so much distended as it was some months before.

On the 15th December he was looking quite fat and comfortable. The abdomen was flaccid, flattened in front, and prominent at the sides, especially on the right. A resistant

mass, dull on percussion, was felt on firm pressure before and behind, in the right lumbar region. The largest circumference of the abdomen was twenty-four inches.

His urine was of a light lemon colour, with a copious deposit of pus and triple phosphates, and was very offensive. After filtration, boiling, and the addition of nitric acid, there was no deposit, notwithstanding the presence of a large quantity of pus. This circumstance, namely, the absence of any indication of albumen by heat and nitric acid, I have also observed in urine which contained numerous blood-discs. It must be explained by supposing that the albumen is prevented from precipitation by some peculiar ingredient; or can it be that pus- and blood-cells may be found without any albumen, except what the cells themselves contain?

For the examination of the urine and many of the clinical observations in this case I am indebted to Mr. S. Gee, M.B., Assistant-Physician and Registrar to the Hospital for Sick Children.

Several cases are on record in which the ureter has been obstructed from birth, and has led to a dilated condition of the pelvis of the kidney. Most of these cases have died young, and the kidney has not attained any large size.

A remarkable case is recorded by Rayer ('Traité des Maladies des Reins,' vol. iii), in which on the right side of the abdomen there was found an enormous cyst, having the anatomical relations of a kidney. This patient lived to the age of twenty-three years. The contents of the cyst were not analysed, so that we do not know whether it contained urinary ingredients.

Another curious case is reported by Rayer, of a youth who lived to the age of seventeen years, and was found after death to present double hydronephrosis; that on the left side being due to a valvular obstruction of the ureter, not completely cutting off communication between the kidney and bladder, and that on the right side being due to a stricture of the ureter.

In the case which I have reported there is obviously a cyst in which the renal secretion accumulates; this cyst does not

usually communicate freely with the bladder, but from time to time, when much distended, its contents are partially poured into that viscus; there is some renal glandular tissue existing on that side, as proved by the constant presence of large quantities of urea in the fluid; the cyst is probably sacculated, and there is not a free communication between all the parts of the cyst, as proved by the difficulty of completely emptying the cyst and of keeping open any fistula which is made. It would seem as if the loculus into which the canula passes is after a time shut off from the rest of the cyst.

Why was no ferrocyanide detected in the fluid of the cyst when it was present in the urine?

Thinking it possible that the presence of albumen in the cystic fluid might prevent the reaction of the ferrocyanide, I gave this salt to another patient who had albuminuria, and failed to detect it in his urine after he had taken twenty grains of the ferrocyanide of potassium.

This induced me to try the same experiment with gallic acid. I found that gallic acid could be detected in his urine after it had become albuminous, but no trace of it was found in the cystic fluid.

This shows either that transudation of gallic acid did not take place through the kidney of the right side, though it did on the left, or else that there was not a free communication between the part of the cyst in which the canula was inserted and that part into which the kidney first emptied itself.

The history of the case clearly proves that the ureter on the right side was often obstructed, but at other times allowed fluid to pass through it.

A valvular obstruction such as existed in one of Rayer's cases or in Dr. Broadbent's case, presently to be mentioned, may be present, and it is possible that the valve may allow fluid to pass under certain conditions, and not under others.

A calculus might possibly be so placed as to cause an intermitting obstruction to the flow of urine down the ureter. I have not met with the record of any case in which a

calculus was so situated as to dilate the kidney to such an extent as in this patient. The absence of pain is entirely opposed to this supposition.

The treatment adopted in this case has been of no service, although it would seem to be the course which the symptoms indicated as suitable. The distension was so great as seriously to endanger the boy's life, as well as to make his life miserable. When the diagnosis was once correctly made, it was obvious that tapping was the only remedy offering any chance of getting rid of the fluid; and inasmuch as the cyst rapidly refilled, the establishment of a permanent fistula appeared desirable, to prevent the necessity for repeated operations. This, however, was found to be impracticable, from the fluid after a short time ceasing to flow. The question was then raised whether the injection of the cyst with iodine was allowable. It was decided that this would be imprudent, seeing that there appeared to be a secreting kidney still existing, that the cavity was so large, and that there would be great risk of inflaming the peritoneum. The next point to consider was whether removal of the cyst as a whole might be attempted. It was thought that the cyst might be removed from behind. Would the risk be greater than that attending the operation of ovariotomy? The left kidney would, no doubt, be able to do the work of two kidneys. There is reason to believe that it is already hypertrophied, and does more than its share of duty. There would, however, I believe, be too great risk attending the operation for extirpation of the cyst, from interference with the sympathetic nerve near the semilunar ganglia, as well as from hæmorrhage and peritonitis. The removal of a kidney from the lower animals has, I believe, always proved fatal.

At present it seems probable that the patient may live for some years in comparative comfort, although much incommoded by the size of his abdomen, and liable at any time to greater accumulation of fluid. Paracentesis may from time to time be necessary, but should only be attempted in cases of extreme distension, seeing that nature usually provides a remedy when she is left to herself. The left kidney is very

likely to suffer from sympathy and from the extra duty thrown upon it.

The irritation set up in the cyst by tapping, and the exhaustion accompanying it, would soon have put an end to his life had the operations been much oftener repeated. It is a very remarkable feature in this case that after such long continued pressure on the right kidney from accumulating fluid there should be left any renal structure capable of secreting urea.

The arteries of this little patient have undergone a peculiar degeneration, such as is usually seen only in old people. Most of the superficial arteries are very tortuous and visible, apparently from a loss of elasticity.

In new-born children hydronephrosis of one kidney has been several times observed.

Cases are reported by Bonet, in the 'Sepulcretum,' vol. ii, p. 290; by Billard, in his 'Traité des Maladies des Nouveaunés,' p. 434; and by Dr. Harley, in the 'Pathological Society's Transactions,' vol. xv, p. 146.

Double hydronephrosis in new-born children is very rare. A case was exhibited at the Académie de Médecine, in 1828, by M. Moreau; and a case due to obliteration of the urethra is reported by Billard, 'Traité des Maladies des Nouveaunés,' page 436.

A specimen was recently exhibited to the Pathological Society by Dr. Broadbent, and reported on by Dr. Harley, in which the ureters were "irregularly distended, so that when filled with water they looked almost sacculated. Water injected from below flowed into the pelvis of each kidney—into the left with facility, into the right with difficulty. The pelvis of each kidney was much dilated, the right twice as much as the left; the former might hold seven or eight, the latter four, ounces of fluid. The parenchyma of the kidney was altered in form and condensed. No calculi or deposit of any kind was detected." The greater distension of the right kidney was found to be due to a circular fold of mucous membrane near the renal end of the right ureter, which acted as a valve and completely blocked up the passage until the

ureter was fully dilated. Several imperfect valvular folds existed in both ureters. The bladder was dilated, but the urethra was not examined. This specimen was taken from a child three months old. The dilatation of the kidneys was, no doubt, congenital.

Cases of pyelitic distension in adults are not very uncommon. Accumulation takes place from temporary obstruction of the ureter, which is repeated at shorter or longer intervals. The following case came under my notice in November, 1855:

A married woman, æt. 47, had suffered for three or four years with pain in the right side of the abdomen, returning at irregular intervals. She had noticed, when the pain was most severe, an oval swelling in the right lumbar region, which disappeared as the pain subsided. She also stated that during the height of the attack her urine was very scanty, but afterwards it was very abundant and very turbid for some days.

When I first saw her, she was in great pain, her pulse was weak, and her extremities were cold. There was much tenderness in the loins, especially on the right side, and in the anterior lumbar region was felt an oval mass, quite dull on percussion, and offering an obscure sense of fluctuation on palpation. She had passed no urine for several hours. She continued to get worse for three days, not passing more than a teaspoonful of water daily. She was delirious, her tongue was yellowish, her pulse very small, and she was drowsy. On the third day she passed a pint of water. On the fourth day there was retention of urine, and three pints of water were drawn off by the catheter. The water passed on the first occasion was not seen, being mixed with menstrual fluid. The urine drawn off by the catheter was light-coloured, acid, containing a small quantity of albumen, and of specific gravity 1013. She soon recovered her usual health, and since that time has had occasional recurrences of pain and increased swelling in the lumbar region; during these attacks her urine has been scanty, but has been passed in unusually large quantities as the pain has subsided.

She has not again had entire suppression of urine since 1855.

Mr. Joseph Thomson reports a case of hydronephrosis in an adult, who was several times tapped ('Path. Society's Transactions,' vol. xiii, page 128).

ON THE

SOLVENT TREATMENT OF URINARY CALCULI;

AN EXPERIMENTAL AND CLINICAL INQUIRY.

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THE possibility of dissolving urinary concretions, or any class of them, within the living passages, is a question of practical interest for the treatment of renal, and, in a less degree, of vesical calculi.

A solvent treatment of these affections cannot, at the present day, be said to have a standing-place in therapeutics. Vesical calculi are made over to the surgeon as soon as they are discovered, to be dealt with by mechanical means, and scarcely any purpose of dealing radically with renal calculi can be said to exist. The opinion expressed by Marcet in 1819 is still the belief of the profession, namely, that "no reasonable expectation can be entertained that calculi lodged

in the urinary organs, and already too large to be discharged by the natural passages, can be actually dissolved by any mode of internal treatment. The only benefit which we may with any confidence expect from medicine in this disease is either to prevent the increase of calculi already formed, or, what is still more important, to guard the constitution of those who are subject to the disorder against the prevalence of the particular diathesis from which it arises."[1]

More recent writers on the subject (Civiale, Rees, Heller) express themselves as doubtfully about solvent remedies; and the small doses which they recommend, indicate very clearly, that the most they expect from their operation is to facilitate the spontaneous expulsion of the concretions, to prevent their further growth, and to palliate their symptoms.

Nevertheless, the past history of the subject contains some convincing proofs that calculi have been dissolved within the bladder by means of alkaline substances; and it appeared worthy of inquiry whether a more exact re-examination of the question might not reveal the causes of the discrepant results obtained in past times, and indicate, with some approach to certainty, what may be rationally expected from a solvent treatment, the cases in which it is applicable, and the precise mode of carrying it out effectually.

With this view, numerous experiments and observations were made by the author at intervals extending over several years; and the results obtained seem to demand a considerable modification of the prevailing opinion. They do not by any means indicate the general possibility of substituting a solvent for a mechanical treatment of vesical calculi, but they suggest an essential improvement in the treatment of renal concretions; they also indicate that uric acid and cystin, under certain circumstances, are capable of solution in the bladder by means of medicines administered by the mouth, at a rate which admits of practical application, and that in certain picked cases a solvent treatment deserves to

[1] Marcet, 'On Calculous Disorders,' p. 143.

be resolutely tried before having recourse to the more dangerous methods of lithotomy and lithotrity.

It may be stated at the outset that the inquiry has not led to the discovery of any new solvent for the stone, but rather to a better application of a treatment which has long been known in principle, but which has forfeited the confidence of the profession by the faulty manner in which it has been carried out, and its employment in cases where it was sure to fail.

For the present purpose urinary concretions may be considered as consisting of *uric acid, oxalate of lime,* and the *earthy phosphates.* Of the rarer varieties, cystin and the urates fall into the same category with uric acid, and carbonate of lime into the same category with the phosphates.

The first part of this paper is devoted to experiments and observations relating to the solvent treatment of uric acid, by alkalizing the urine, through the internal administration of medicines. The second part is devoted to experiments relating to the solvent treatment of uric acid by injections into the bladder, and to experiments on the solvent treatment of oxalate-of-lime and phosphatic concretions.

With regard to uric acid the inquiry is tolerably complete in the directions taken. Oxalate of lime gave results which did not promise any useful application. Sir B. Brodie's method of dissolving phosphatic calculi by injecting acid solutions into the bladder was imitated in one experiment with results highly favorable to further trial in the same direction.

PART I.

OBSERVATIONS AND EXPERIMENTS RELATING TO THE SOLVENT TREATMENT OF URIC-ACID CALCULI, BY ALKALIZING THE URINE, THROUGH THE INTERNAL ADMINISTRATION OF MEDICINES.

The idea of imparting solvent properties to the urine by means of medicines taken internally is of very ancient date, and a multitude of substances have attained, at different times, a temporary reputation as solvents for the stone. But there is no reason to believe that any of them possess real virtues except those which are capable of rendering the urine alkaline.

It has now been known for many years that the fixed alkalies, in the caustic state, or as carbonates, citrates, acetates, or tartrates, when administered by the mouth, appear in the urine as carbonates, and thus render the urine alkaline.

It has also been asserted, and on the other hand has been denied, that the alkaline carbonates exercise a solvent action on uric acid.

This power of alkalizing the urine at will, has been hitherto very imperfectly studied in its application to the solvent treatment of urinary calculi. We have no information as to the degree of alkalescence which can be communicated to the urine by given doses of any salt, nor as to the relative alkalizing power of different salts. We are also ignorant of the relative solvent power on uric acid of carbonate of potash and carbonate of soda; we do not know the best strength of solution to employ, nor the effect of quantity of the solution on the results obtained, nor the absolute rate of dissolution attainable. Only the rudest notions prevail of the actual effect of alkalized urine on calculi in the bladder, and of the collateral circumstances which favour or impede its operation.

An extended inquiry was found necessary to elucidate all these points, including a long series of experiments in the laboratory, numerous observations on healthy persons, and clinical observations on patients suffering from renal or vesical calculi.

These experiments and observations are detailed in the following pages in the above order. An experimental basis is first established, so far as is practicable, then the clinical facts are added, and finally the rules for conducting the solvent treatment[1] are enunciated.

Tentative experiments soon showed that solutions of the carbonate of potash and carbonate of soda did exercise a solvent action on uric acid, but only within certain limits of strength.

The questions which first presented themselves for decision were:—the relative solvent powers on uric acid of carbonate of potash and carbonate of soda, the relative effects of solutions of different strength, and of varying quantities of solutions of the same strength, also the absolute rate at which dissolution proceeded under conditions which might be more or less closely imitated within the body of a patient.

Answers to these questions were sought by placing sections of uric-acid calculi, weighing about 100 grains, in ten-ounce phials, and causing currents of the different solutions to pass over them at a regulated rate. The plan adopted was to immerse the phials in a water bath, kept constantly at the temperature of the body. Each phial was connected on the one side by means of india-rubber tubing with a reservoir of the solution to be tried. On the other side an exit-tube was provided, which was fitted at its extremity to a piece of glass tube drawn to a fine point. The bore of this tube at the point regulated the rate of flow. The flow of the solution over the stone was maintained, in a large number of experiments, at the rate of six pints in the twenty-four

[1] Whenever the term "solvent treatment" is used in the first part of this paper it signifies exclusively the method of treating uric-acid calculi by alkalizing the urine by the administration of medicines by the mouth.

hours; in some experiments it was increased to eight, ten, and fifteen pints; in others diminished to three pints or less. The experiments were contrived so as to imitate, in as close a manner as possible, the gentle but incessant flow of the urine along the ureters.

Section I.—*Comparison of solutions of carb. potash and solutions of carb. soda.*

The potash salt was found sensibly to excel the soda salt as a solvent for uric acid. When the granular carbonate of potash of the shops was compared with the dried carbonate of soda, the advantage lay with the former when equal weights were employed, and still more when corresponding atoms were compared. Solutions of these two salts yielded the following results:

A solution of *carbonate of potash* containing 30 grains to the pint dissolved daily (mean of twelve day-experiments[1])	11·9 per cent.
A solution of *carbonate of soda* containing 30 grains to the pint dissolved daily (mean of eight day-experiments)	10·3 „
A solution of *carbonate of potash* containing 60 grains to the pint dissolved daily	20·2 „
A solution of *carbonate of soda* containing 60 grains to the pint dissolved daily	14·3 „

Carbonate of potash possessed a further advantage over carbonate of soda in its *wider range* of solvent power with the stronger solutions. This point will be better understood after the effects of solutions of different strength have been considered in the following section.[2]

[1] By day-experiments are meant experiments continued for twenty-four hours.

[2] Some experiments were also made with carbonate of lithia, which has been vaunted in recent times as a solvent for uric acid. Its power was found much inferior to that of the carbonates of potash and soda. Its reputation seems to have been gained through its comparative insolubility. Only weak solutions of it *could* be employed, and then were com-

SECTION II.—*Comparison of solutions of different strength.*

The strength of the solution was found to affect its solvent capacity more than any other condition. The greatest solvent power was found to lie in solutions containing from forty to sixty grains of carbonate to the imperial pint. With stronger solutions than these the fragments became covered over with a white crust of alkaline bi-urate, which seriously interfered with the solvent action.

A solution of carbonate of soda containing 120 *grains to the pint* caused the production of a stiff coating of bi-urate, resembling white paint, which altogether prevented any dissolution taking place. The fragment, after undergoing the effect of such a solution for twenty-four hours, had not lost any weight; and when the white coating had been scraped off with the nail the loss of weight was only three quarters of a grain.

A solution of carbonate of potash containing 120 *grains to the pint* also produced a crust of bi-urate, but of a much looser texture. The fragment had lost weight to the extent of 3 or 4 per cent. in the twenty-four hours, and when the crust was scraped off the loss amounted to from 6 to 11 per cent.

With solutions containing 160 *and* 240 *grains to the pint* there was no loss of weight with potash or soda; the fragments were invested with a thin tough coating of white bi-urate, which put a stop to all solvent action.[1]

With solutions of carbonate of potash containing 80 *grains to the pint* the fragment was found covered with a loose detachable crust of bi-urate, like a layer of whitewash, which chipped off readily in large flakes or scales when touched

pared with solutions of potash and soda which were much too strong for effective dissolution. The lithia experiments will be found recorded in the second part of this paper.

[1] For the analysis and further particulars of the white coating of bi-urate, see an extract of a paper by the author in the 'Transactions of the British Association for the Advancement of Science' for 1861; also in 'Beale's Archives' for 1862.

with the nail. Two experiments, each of twenty-four hours' duration, made with a solution of this strength, showed a mean dissolution of 9·8 per cent. when the fragments were weighed without detaching the loose crusts. After detaching the crusts the loss of weight amounted to 13 per cent.

With a solution of carbonate of potash of 60 *grains to the pint* the fragment was usually dissolved clean, without a trace of bi-urate crust; but in some experiments detached scales of bi-urate were found on the spots where the stone had rested in the phial. Dissolution went forward with great rapidity with solutions of this strength, and the production of the loose crusts did not appear to interfere sensibly therewith. Seven day-experiments with this solution dissolved the fragments at a mean daily rate of 20·2 per cent. of their weight.

Solutions of carbonate of soda of the same strength (60 grains to the pint) showed a complete and more adherent crust of bi-urate than the corresponding potash solution. The mean loss of weight in two day-experiments, after detaching the crust, amounted to 14·3 per cent.

Weaker solutions than 60 grains to the pint dissolved the fragments without the formation of bi-urate crusts, or only with traces of such; the red surface of uric acid remained clean and appeared water-worn. The dissolving powers declined gradually as still weaker solutions were used.

Solutions of carbonate of potash gave the following results:

				Per cent.
40	*grains to the pint*		(daily flow, six pints—mean of three experiments) dissolved in twenty-four hours	15·6
30	„	„	(daily flow, six pints—mean of three experiments) dissolved in twenty-four hours	11·9
20	„	„	(daily flow, six pints—mean of three experiments) dissolved in twenty-four hours	11·0
10	„	„	(daily flow, six pints—mean of three experiments) dissolved in twenty-four hours	6·5

	Per cent.
5 *grains to the pint* (daily flow, eight pints—mean of two experiments) dissolved in twenty-four hours	6·0
2½ „ „ (daily flow, eight pints—mean of two experiments) dissolved in twenty-four hours	2·8
1 „ „ (daily flow, eight pints—mean of two experiments) dissolved in twenty-four hours	1·2

The effect of dilution was further tried in a second set of experiments, in which the flow of the solution was reduced to between one and two pints per day. The results stand as follows, with solutions of carbonate of potash:

	Per cent.
40 *grains to the pint* (mean of four experiments) dissolved in twenty-four hours	17·1
20 „ „ dissolved in twenty-four hours	7·0
10 „ „ „ „	5·0
7 „ „ „ „	6·6
6 „ „ „ „	5·8
5 „ „ „ „	3·7
4 „ „ „ „	2·9
3 „ „ „ „	1·3
2 „ „ „ „	0·7
1 „ „ „ „	1·0

From the two sets of experiments we gather that solutions containing less than 3 grains to the pint exercise only a feeble solvent action on uric acid. The Manchester pipe-water was found to possess a solvent power not greatly inferior to this. In an experiment extending over seven days, the pipe-water, flowing at the rate of nine pints a day, at blood heat, dissolved 5 grains from a fragment of uric acid weighing 112 grains, or at the rate of ·6 per cent. in twenty-four hours.

Solutions containing from 4 to 10 grains to the pint had a sensibly greater effect. The daily rate of dissolution with solutions of this strength ranged from about 3 to about 6 per cent. of the weight of the fragment.

Solutions of 20 grains to the pint and upward, as high as

60 grains to the pint, displayed a still more powerful action. They dissolved the fragments at daily rates varying from 7 per cent. to more than 20 per cent.

SECTION III.—*Comparison of the effects of varying quantities of solutions of constant strength.*

The natural flow of urine in healthy persons varies from two to three pints a day, but by copious water-drinking the quantity may be readily increased to six or ten pints a day. It was, at the outset of the inquiry, thought that the quantity of the solvent permitted to flow over the stone would greatly influence the rate of dissolution, but when the point was tested experimentally the effect of quantity, between the limits necessarily imposed by the capacity of the kidneys to eliminate aqueous fluids, proved to be comparatively unimportant.

In order to obtain comparable results, different quantities of a solution of constant strength were passed over the same stone on successive days.[1]

A solution of carbonate of soda containing 30 grains to the pint gave the following results:

	Per cent.
With a daily flow of 15 *pints* the loss of weight was (mean of four experiments)	10·9
With a daily flow of 8 *pints* the loss of weight was (mean of four experiments)	10·2
With a daily flow of 5 *pints* the loss of weight was (mean of two experiments)	9·8

A solution of carbonate of potash containing 30 grains to the pint gave the following results:

[1] Absolutely exact comparative results are necessarily impossible in experiments of this kind. Uric-acid stones differ considerably among themselves in density of structure, and the fragments vary in form, weight, and surface, so that some offer considerably greater facilities for solution than others. The experiments were, however, repeated a sufficient number of times to cover discrepancies from these sources. The general conclusions may be confidently relied on.

	Per cent.
With a daily flow of 15 *pints* the loss of weight was (mean of four experiments)	13·0
With a daily flow of 8 *pints* the loss of weight was (mean of two experiments)	15·0
With a daily flow of 6 *pints* the loss of weight was (mean of four experiments)	10·2
With a daily flow of 4 *pints* the loss of weight was (mean of two experiments)	9·5

A solution of carbonate of potash containing 60 grains to the pint yielded the following results:

	Per cent.
With a daily flow of 14 *pints* the loss of weight was (mean of two experiments)	19·0
With a daily flow of 6 *pints* the loss of weight was (mean of two experiments)	21·4

A number of other experiments, which it is needless here to reproduce, pointed to the same deduction, namely, *that with solutions of constant strength a flow of eight to fifteen pints a day scarcely dissolved more than a flow of three to six pints a day.*

The inquiry, so far, has led us to the following conclusions:

a. Carbonate of potash is a better solvent for uric acid than carbonate of soda.

b. The maximum solvent power of the alkaline carbonates on uric acid lies in solutions containing from 40 to 60 grains to the pint.

c. That within the limits prescribed the effect of quantity of the solution is unimportant.

SECTION IV.—*Absolute rate of dissolution of uric-acid calculi in solutions of the alkaline carbonates.*

With regard to the absolute rate of dissolution, the experiments above detailed opened out an inviting prospect. The solutions of maximum solvent power dissolved from 10 to 20 per cent. of the weight of the stones in the course of twenty-four hours. If results approaching these, or one

half, or even one quarter, could be obtained within the living body, a little consideration will show that such an impression could be made on the concretion in a few weeks, as would either entirely dissolve it, or reduce its dimensions to a point which would enable it to escape by spontaneous expulsion.

The next points to be ascertained were—the best way of alkalizing the urine so as to impart to it an alkalescence corresponding to that of solutions of carbonate of potash in water of maximum solvent power; also to examine the actual effect of alkalized urine passed over uric-acid calculi in a phial at blood heat.

SECTION V.—*The most convenient way of alkalizing the urine, the degree of alkalescence which can be communicated to it, and the doses required to produce the desired effect.*

The urine may be rendered alkaline by a variety of substances; but the best adapted for the purpose are the alkaline bicarbonates, acetates, and citrates. These salts have very little taste, they do not disturb the stomach and bowels, and they may be administered for weeks and months without deleterious effects. All of them appear in the urine as carbonates of their respective bases.

After a large number of comparative trials the acetate and citrate of potash were found to offer advantages superior to any of the others. They form draughts which are nearly tasteless; being neutral salts, they do not saturate the free acids of the stomach, and thereby interfere with digestion; they are also extremely soluble.

The degree of alkalescence communicated to the urine by these salts depends on the quantity and dilution of the dose.

To maintain a persistent alkalescence of the urine by acetate or citrate of potash, required, in an adult, six or eight drachms to be given daily, in divided doses. When administered in these large doses about two thirds of the potash reappeared in the urine as free carbonate.

When the acetate or citrate of potash, in the dose of 40 grains dissolved in 5 ounces of water, was administered every two hours, it communicated to the urine an alkalescence, equivalent to that of a solution of carbonate of potash containing from 20 to 80 grains to the pint.[1] This corresponds sufficiently closely with the maximum solvent power of that salt as already ascertained. (See Section II.)

It is, of course, quite impossible to maintain the urine at a constant degree of alkalescence, however short may be the intervals at which the medicine is administered. The activity of the kidneys oscillates from hour to hour; at one time the urine is secreted abundantly and highly dilute, and then the degree of alkalescence falls; at another time it is secreted more scantily and in more concentrated form, and then the degree of alkalescence rises. When, however, the dose is exhibited with regularity, at intervals of two or three hours, the oscillations rarely pass the limits above indicated, namely, an equivalent to 20 to 80 grains of carbonate of potash to the pint; and, as a rule, the alkalescence ranges between 35 and 60 grains of carbonate to the pint.

The *tartrates* were found unsuitable, from their bitter taste, and their tendency, unless greatly diluted, to act on the bowels.

The *common phosphate of soda* and the *basic phosphate of soda* were found to possess very inferior alkalizing powers. The former, even in the quantity of 640 grains daily, in divided doses, only produced a total daily alkalescence equal to 34 grains of carbonate of potash; and the subphosphate, in the same quantity, only a total alkalescence equal to 48 grains of carbonate of potash per day; whereas the same quantity of citrate produced a total daily alkalescence of more than 300 grains of carbonate of potash.

[1] The degree of alkalescence of the urine was estimated by volumetrical analysis with a standard solution of dilute sulphuric acid. Whenever carbonate of potash is referred to in this paper as a standard of alkalescence, the granular carbonate of potash of the shops is signified. This preparation contains, according to Phillips, 16 per cent. of water, and its combining equivalent is reckoned as 82·5.

Borax proved objectionable on account of its unpleasant effects on the stomach and its feeble alkalizing powers. With 320 grains of borax a day, which was difficultly borne by the stomach, the total alkalescence produced in twenty-four hours was only equal to 14 grains of carbonate of potash.

These observations on the phosphates of soda and borax were made on eight different individuals, and extended in each case over several successive days.[1]

Section VI.—*The effect of alkalized urine on uric-acid calculi.*

Having ascertained the method of alkalizing the urine to a degree corresponding with the maximum solvent power of solutions of carbonate of potash in water, an experiment was now performed imitating still more closely actual clinical experience. This consisted in subjecting uric-acid calculi to the operation of alkalized urine in a phial at the temperature of the body.

Some practical difficulties were encountered in performing this experiment. It was found impossible to maintain a constant current over the stone, on account of the clogging of the tubes; and a simpler method had to be adopted. This was to transfer the alkalized urine, immediately it had been voided, into a ten-ounce phial containing a uric-acid calculus. The phial and its contents were kept in a water bath at blood heat. Each time the bladder was emptied the old urine was thrown away and replaced by the fresh urine.

But even when thus simplified, certain precautions were found essential to the successful performance of the experiment. And as these precautions depend on a circumstance of great importance to the efficient carrying out of the solvent treatment in practice, a few explanations are here necessary.

It is well known that fresh urine has a tendency to become quickly ammoniacal when exposed to the contact of stale or decomposed urine; this is especially the case when

[1] In the second part of this paper will be found some observations on the solvent power on uric acid of watery solutions of these salts.

the urine is re-warmed in the air. Alkalized urine exhibits this tendency more strongly than acid urine. In my earlier trials this tendency came into play, and caused some embarrassment; eventually it led to a valuable practical deduction, as may be seen from the following observation.

Alkalized urine from a person taking bicarbonate of potash was placed in a phial with a fragment of uric acid, and maintained at blood heat. The urine was changed, according to the frequency of micturition, every two or three hours. At the end of twenty-four hours the fragment was found to have an offensive smell, and to be covered in great part with whitish patches. It had diminished in weight from 78 to 76 grains. The experiment was continued for thirty-six hours longer, and the fragment re-examined. It was now covered with a complete white crust; it was very offensive and ammoniacal to the smell; its loss of weight was almost inappreciable until after the white crust had been scraped off. The phial in which the experiment was performed was also encrusted with a whitish substance of similar character. The crust on the fragment and the deposit on the sides of the phial were found on examination to be composed of a mixture of triple phosphate and amorphous phosphate of lime; in other words, of "fusible matter" or "secondary" calculous deposit.

In a second experiment, performed similarly, the phosphatic crust was estimated to be a tenth of a line thick; the fragment entirely ceased to lose weight after the second day.

From these experiments the important deduction was drawn—*that in an ammoniacal urine the solvent power of alkalized urine is completely nullified by the deposition of the mixed phosphates on the surface of the concretion.*

In subsequent experiments the following simple expedient was adopted, which obviated this difficulty. At each change of urine the fragment was dipped into cold water, so as to remove all traces of the previous urine; the phial, in like manner, was thoroughly cleansed at each remove. By this means the fragment and phial were kept perfectly sweet and free from the contamination of decomposing urine.

Performed in this manner, the experiment succeeded perfectly; not a particle of phosphate was deposited, the surface of the fragment remained clean and red, and dissolution went on uninterruptedly.

In two such experiments, each of twelve hours' duration, a section of uric-acid calculus weighing 180 grains lost weight to the extent of 6 grains in one and 6½ in the other, or at the rate of 12½ grains in the twenty-four hours. As absolute values, these numbers are necessarily too low. At each change of urine a certain time was lost, and the urine-solvent was thereby allowed to cool to a certain degree; a further temporary lowering of the temperature was occasioned by the washing of the fragment and phial in cold water.

The following tables exhibit the condition of the urine during the performance of these two experiments—showing its varying quantity, specific gravity, and alkalescence, at each micturition.

The individual whose urine was employed was fully under the influence of citrate of potash in the dose of 40 grains, administered every two hours, in 5 ounces of water.

First Experiment.—The immersion of the fragment commenced at 9.40 a.m., and terminated at 9.40 p.m. The urine used was secreted between 7.30 a.m. and 8.8 p.m.; it was transparent throughout. The fragment weighed before the experiment 180½ grains; after the experiment 174 grains. Breakfast at 8.40 a.m.; dinner at 1 p.m.; tea at 5 p.m.

Time during which the urine was secreted.	Quantity of the urine.	Sp. gravity.	Alkalescence per pint in terms of pot. carb. of shops.
7.30 to 9.36 a.m.	6 oz.	1022	65·0 grains.
9.36 to 10.50 „	15¼ „	1008	26·9 „
10.50 to 12.20 p.m.	8 „	1012	35·5 „
12.20 to 2.3 „	9 „	1013	43·6 „
2.3 to 3.15 „	12¼ „	1009	35·5 „
3.15 to 4.45 „	9 „	1013	49·8 „
4.45 to 5.57 „	6¾ „	1013	46·7 „
5.57 to 6.45 „	7 „	?	17·7 „
6.45 to 8.8 „	10¾ „	1008	20·8 „

Second Experiment.—The immersion of the fragment commenced at 9.30 a.m., and terminated at 9.30 p.m. Its weight before the experiment was 174 grains; after the experiment 168 grains. The urine employed was secreted between 7.37 a.m. and 9 p.m.; it was perfectly clear throughout. Breakfast at 7.40; dinner at 1.40; tea at 5 p.m.

Time during which the urine was secreted.	Quantity.	Sp. gravity.	Alkalescence per pint in terms of carbonate of potash of the shops.
7.37 to 9.30 a.m.	3½ oz.	1025	82·2 grains.
9.30 to 10.50 „	12 „	1008	30·4 „
10.50 to 1.30 p.m.	9¾ „	1015	58·4 „
1.30 to 4.20 „	9¼ „	1013	48·0 „
4.20 to 5.55 „	5½ „	1018	72·0 „
5.55 to 7.10 „	10 „	1006	26·9 „
7.10 to 9.0 „	5 „	1016	59·9 „

The urine of patients taking full doses of acetate or citrate of potash is generally clear, as in the above experiments, and shows no tendency to precipitate even on standing. But this is not invariably the case; it is sometimes turbid from deposition of the amorphous phosphate of lime. Two conditions seem especially to favour this deposition, namely, the febrile state and the digestion of a heavy meal. The amorphous phosphate is not infrequently deposited after a meal in healthy persons who are not taking any alkalizing medicines; the circumstance is therefore not to be regarded as an unnatural or hazardous one.[1]

The amorphous phosphate of lime differs essentially from the "mixed phosphates" or "fusible matter" thrown down in an ammoniacal urine. The former is a loose flocculent substance, which shows no tendency to aggregate into concretions; the latter, on the other hand, is partly crystalline, and speedily encrusts any object brought into contact with it. The establishment of this distinction disposes of one objection which has been urged against the use of alkaline solvents.

[1] See a paper by the author "On the Daily Changes of the Urine," in the 'Edinburgh Med. Journ.' for March and April, 1860.

The inquiry has now brought us to the following additional conclusions:

d. Urine can be readily alkalized to a mean degree corresponding with the maximum solvent power of a solution of carbonate of potash in water.

e. Urine so alkalized, maintained at the temperature of the body, in contact with a uric-acid calculus, was found, on actual trial, to dissolve the concretion at the rate of 12 or 13 grains in the twenty-four hours.

f. The oscillations in the degree of alkalescence of the urine did not appear to exercise any unfavorable influence on its solvent power.

g. The fragment experimented on showed no tendency to contract a coating of bi-urate or of earthy phosphate.

It now remains to bring forward illustrations of the application of the solvent treatment in actual practice; to distinguish the cases in which the treatment is applicable from those in which it is inapplicable; to lay down directions for carrying it out effectually; and, finally, to examine some of the objections which have been urged against its employment.

Section VII.—*Illustrations of the application of the solvent treatment in practice.*

A.—*Cases of renal calculi and gravel.*

One of the first rational attempts to treat renal gravel of uric acid by the alkaline carbonates was made by the celebrated Mascagni on his own person. He gives the following account of his case in the 'Memoirs of the Italian Society' for 1804:

"I had been subject for several years to pains in the lumbar regions, and I voided from time to time gravelly concretions of a yellow-ochre and brick-red colour. Knowing that gaseous alkaline fluids had been used in such cases, I took some on several occasions with benefit. I imagined I could get greater effects with carbonate of potash.

"In the months of August and September, 1799, having

been obliged to lead a sedentary life, I was cruelly attacked with pains in the kidneys, and I voided a considerable number of small concretions, some of which were large enough to be regarded as veritable calculi. They were reddish and crystalline, they were deposited at the bottom of the vessel each time I made water, and I could see their glistening facets through the transparent urine. I was also subject to an excess of acid in the stomach, which was perceived in the mouth. I examined my urine and found in it a free acid, which, as well as the concretions, I recognised as consisting of uric acid.

"Having thus assured myself of the nature of the concretions I was voiding, I resolved to make use of the carbonate of potash and to observe the result. I took the first day about a drachm, one half in the morning fasting, and the other half in the evening. I dined at one o'clock in the afternoon. This salt, dissolved in ten ounces of water, had very little taste, it caused no disturbance of the stomach or bowels; but as soon as I swallowed it, it occasioned a considerable disengagement of carbonic acid gas, which was felt in the mouth and discharged by the anus.

"The second day I took two drachms, and the third day three drachms; and I continued this dose, dissolved in twenty ounces of water, for ten days. Before using the carbonate my urine was very acid, and intensely reddened blue litmus paper. On the second day the paper changed colour very little, and none at all on the third day. The acid of my urine was therefore saturated. At this time the renal pains diminished, and I voided no more gravel with the urine. Afterwards the pains ceased entirely, the urine became less loaded, and *I recognised the potash in excess.*

"I ceased to use the carbonate of potash, and for months I voided no concretions. Being subsequently attacked by the same symptoms, I had recourse to the same remedy, and had the same good results. I have repeated this medico-chemical experiment every time I have felt the same inconvenience, and always with success. Two years have now

elapsed since I voided any concretions, though I no longer make use of the potash."[1]

Cases resembling this might be readily multiplied; but their evidence in favour of dissolution having positively occurred, falls short, from the nature of the circumstances, of rigorous demonstration. I will only add to it one which occurred in my own practice.

In July, 1860, a stout middle-aged gentleman brought me eleven small concretions, varying in size from a large pin's head to a pea. He had voided these with the urine a few days previously; they were composed of nearly pure uric acid. He stated that three years before, he was attacked with severe renal colic, which subsided on the third day with the discharge of a small calculus by the urethra. From this period to the time of my seeing him attacks of renal colic, terminating in the discharge of small brownish concretions, recurred with great regularity at intervals of three or four months.

The urine was found to be acid and high coloured; the general health was somewhat impaired by his periodical sufferings.

In projecting the plan of treatment it was considered that the patient had in all probability a number of similar concretions still lodged in the kidneys. The dissolution of these was the first object; the next was to prevent their future formation. Seeing the small size of the concretions, it was thought that, by keeping up a persistently alkaline state of the urine for a week or two, complete dissolution of them would be effected. With this view, citrate of potash, in two-scruple doses, dissolved in half a pint of water, was administered every three hours for the space of a fortnight. Afterwards the patient took a drachm of the same salt in a tumbler of water, night and morning, for a period of three months. As no recurrence of the renal pains took place, nor the discharge of any concretions, the medicine was then discontinued; but the patient was instructed to take every

[1] Magendie, 'De la gravelle,' p. 85.

night before going to bed a tumbler and a half of simple water, a practice which he has continued up to the present time (October, 1864). There has been no return of the symptoms.

B.—*Cases of vesical calculi.*

A considerable number of examples of the successful treatment of vesical calculi by alkaline solvents lie buried in the forgotten publications which appeared in this country about the middle of the last century, when the remedy of Miss Stephens made so great a noise.[1] Some fifteen or twenty cases were also collected by Chevallier and Petit when the question was resuscitated in France thirty years ago.

Most of these reports are vitiated by the absence of information as to the nature of the stone and the condition of the urine. At the former epoch (1740) urinary calculi were all supposed to be of one nature, and that an unknown one. At the latter epoch the chemical composition of urinary calculi was, indeed, known, but some of the most important points in their development were misunderstood; urinary chemistry was still in its infancy; and the same absurd pretension of universal efficacy was put forth on behalf of alkaline substances which swamped their reputation in 1740.

One of the best illustrations from the earlier records is supplied by Dr. James Jurin, who was himself the sufferer. He states that he was subject for many years to red gravel. At Christmas, 1740, he voided a small stone, after suffering four days from nephritic colic. In January and February following he perceived unmistakeable signs of stone in the bladder. These he describes at great length and with remarkable clearness.

In March he began to take lixivium of soap or soap-ley (a strong solution of caustic potash), in gradually increasing quantities until he reached the amount of an ounce or an ounce and a quarter daily. He took for a single dose one or two teaspoonfuls of the ley, diluted with three quarters of a pint of water. The soap-ley which he employed was

[1] Ploucquet gives a list of more than forty papers and pamphlets published on the subject about 1740.

"one-fifth part heavier than river water" (*i. e.* its specific gravity was 1200, which is about three times as strong as the liquor potassæ of the London Pharmacopœia).

He continued this treatment for five months. On July 10th he voided a small smooth stone, of the size of an oat and of a reddish colour. On the 27th of the same month he voided a second stone. On August 6th he voided a third stone, and about the beginning of September a fourth. All his symptoms now disappeared, and he discontinued the medicine; but in December he had a return of the vesical symptoms; he also noticed that his urine again furred the chamber-pot, and that he voided a little red gravel, as he had previously done. He went back to the soap-ley, and in the course of a week parted with a small, rough, reddish stone. From this time he continued perfectly easy. He still took a couple of teaspoonfuls of the lixivium each day, and this he found sufficient to keep the urine from furring the utensil.

The concretions in this case were undoubtedly uric acid, as may be learnt, not only from their red colour, but also from an experiment which Dr. Jurin has recorded. He states that they dissolved entirely in the alkaline ley and in lime water.[1]

Of the cases recorded in France I shall only cite one. In Chevallier's essay on 'Solvents for the Stone,'[2] ten cases of the successful use of bicarbonate of soda are described. Dr. Charles Petit[3] has contributed some half-a-dozen additional cases illustrating the effects of Vichy waters (which contain 44 grains of bicarbonate of soda to the pint). The following is one of the latter.

[1] The record of this case may be found bound up with Rutty's 'Observations on Joanna Stephen's Medicine for the Stone,' Lond., 1742. Another good case is related by Whytt, in his 'Essay on Lime-water,' p. 5, Edin., 1752; and a third, in which the successful result is vouched for by a post-mortem examination made seventeen years afterwards, is recorded in the 'Philos. Trans.' for 1757, by Dr. Pringle.

[2] Translated by Edwin Lee in the 'Med. Gazette,' vol. xx, p. 542.

[3] Dr. Ch. Petit, 'Du mode d'action des Eaux Minérales de Vichy,' Paris, 1850.

M. de L—, æt. 51, was sounded by Leroy d'Etiolles, who found a stone in the bladder. This he believed not to be large, and to be suitable for crushing. The patient, however, went to Vichy, and drank, the first day, seven or eight glasses of the waters. The next day he took fifteen, and the urine, which was previously very acid, became strongly and constantly alkaline. In a few days he took twenty-two and twenty-four glasses. The symptoms, which were before severe, now subsided more and more; and after seventeen days of treatment he voided a smooth uric-acid concretion, which bore evident traces of dissolution. From this moment he continued wholly free from symptoms, and was able to take violent equestrian exercise without the least inconvenience.'[1]

Before proceeding to relate my own experience, it may not be amiss to examine shortly into the reasons why the solvent treatment, in spite of a large mass of evidence in its favour, failed to maintain itself, and fell into universal neglect, both after 1740 and more recently after 1840. This question is now not difficult to answer. At both epochs the advocates of solvents claimed to be able to deal successfully with urinary calculi of every kind. It was alleged that alkaline remedies, either by solution or disintegration, could destroy them all, sooner or later. Dr. Petit, indeed, admitted that mulberry and bone-earth calculi were only very slowly acted on; but with regard to the mixed phosphates, he insisted, on the faith of some ill-contrived experiments, that they were easily soluble in the alkaline bicarbonates. The capital fact, announced in a previous page, was then unknown—that in ammoniacal urine the alkaline carbonates are absolutely powerless to dissolve any stone, or even to prevent its increase, in consequence of the continual precipitation of fresh phosphatic deposit on its surface.

The indiscriminate use of the alkaline treatment could only result in disappointment; failures multiplied more rapidly than successes. Some of the individuals whose cases

[1] Ch. Petit, loc. cit., p. 272.

were reported as cures by the Parliamentary commission appointed to examine the effects of Miss Stephens's remedy (soap and calcined egg-shells), were found, when their bodies were opened after death, to contain large stones in their bladders. Civiale and others published cases in which patients had passed from bad to worse under the alkaline treatment, and had at length been reduced to a point at which operative procedures were no longer available. The facts which told against the efficacy of solvents were, from their very nature, more incontestable than those which told in their favour. When a patient had taken the solvent for an adequate period, and yet a stone was afterwards extracted from his bladder by operation, or was found there after death, that was a kind of fact which could not be evaded by any sophistry; whereas explanations were not so difficult to find for the disappearance of the symptoms and signs of a vesical calculus under a course of alkalies, without admitting that dissolution of it had taken place. In the absence of any key to explain the direct contradiction of the evidence on the two sides, opinions settled, as was natural, on the side of the more palpable facts. The reported cures were explained away: either (it was said) the sounding was fallacious, and there never was a stone in the bladder; or, if concretions came away under the use of the medicine, these never had been any larger—their coming away was a mere coincidence, and not a consequence of the treatment.

Nor was the indiscriminate use of the remedy the only cause of its disgrace. We must also take account of the imperfect manner in which the treatment was carried out, and the absence of any reliable data whereon to form an opinion as to the rate or speed at which dissolution could take place in the body. In the earlier epoch alkaline substances were given in the form of soap, calcined egg-shells, lime water, or solutions of caustic potash—all of them nauseous, apt to derange the stomach, and difficult to administer in sufficient doses to prove efficacious. In the second epoch Vichy waters were chiefly relied on. These contain soda, which, as we have seen, is an inferior solvent to potash, and

the great dilution of the remedy in the Vichy waters must have seriously impaired its power.

I now proceed to give an account of my own experience. It was gathered before some important points were properly understood, which later inquiries have made clear to me. My first case was one of uric-acid calculus, and in every way suitable for the solvent treatment; but owing to my want of better knowledge at that time the treatment was carried out very imperfectly, and was not persevered in sufficiently long to effect complete dissolution. In my second case the stone was composed of alternate layers of uric acid and oxalate of lime. The latter substance completely resisted the solvent, while the former was partly worn away. The specimen, from the peculiarity of its structure, offers an interesting and irrefragable proof of the solvent power of alkalized urine on uric-acid concretions. The third patient proved to have a mulberry or oxalate-of-lime calculus. When afterwards extracted by operation it showed not the slightest trace of dissolution.

Case 1.—David F—, æt. 4, a well-grown healthy child, was admitted into the Manchester Royal Infirmary, December 1st, 1858, under the care of Mr. Southam. The patient's mother stated that distinct symptoms of stone (pulling the prepuce, dancing and screaming with micturition) had appeared three years before, and had continued, with interruptions, ever since. On sounding, a stone was detected; the symptoms were quiescent on admission.

By the courtesy of Mr. Southam the case was made over to me for the purpose of trying the effects of solvents. The urine was freely acid, of an amber colour, perfectly natural, except that it contained a few pus-corpuscles. Frequent examination failed to detect any crystalline deposit in the urine, except after long standing, when it let fall some crystals of uric acid. The bladder was not irritable. The condition of the urine indicated that the calculus was composed either of uric acid or of oxalate of lime or of alternating layers of these two substances, but which of the three kinds

there was no evidence to decide.[1] The acid reaction of the urine precluded the idea of any phosphatic concretion.

On December 17th, 20 grains of the tartrate of potash-and-soda (Rochelle salt), dissolved in four ounces of water, were ordered to be taken every two hours. The urine next day was alkaline; its specific gravity varied from 1007 to 1015. The same doses, at the same intervals, were administered regularly without change or interruption for a period of three weeks. The urine, which was frequently examined during this period, amounted generally to three or four pints a day. It was perfectly sweet when voided, and always more or less alkaline; the specific gravity ranged from 1006 to 1016, the night urine being always denser than that of the day.

On January 7th, 1859, the tartrate was increased to 30 grains, dissolved in five ounces of water, every two hours. On January 12th the same dose was directed to be taken in six ounces of water. This treatment was continued until January 28th. On that day the boy was sounded by Mr. Southam, and the stone detected. We thought it unadvisable to persist longer with the treatment; two days after, the patient was cut by Mr. Southam, and two small stones were extracted by the lateral operation. The boy made an excellent recovery.

The condition of the urine in the last three weeks was as follows:—The quantity varied from five to six pints daily; its specific gravity ranged from 1004 to 1007, sometimes it rose to 1011. It was invariably alkaline, but very feebly so. It was always sweet when voided, and quite free from any signs of ammoniacal decomposition.

The two calculi weighed together 22 grains; they were composed of pure uric acid. Their surfaces were devoid of the granular or minutely tuberculated appearance usual on uric-acid calculi, but perfectly smooth and polished, like river

[1] Strictly speaking, the stone might also have been cystin, but the great rarity of this calculus, and the still greater rarity of such a calculus existing without any cystin deposit in the urine, rendered this possibility a very remote one. So far as regards the solvent treatment, cystin falls into the same category with uric acid. (See Appendix to Part I of this paper.)

pebbles, with a finely veined structure. Not a particle of phosphatic deposit existed on their surfaces.

The additional experience which I now possess enables me to see that the treatment in this case was not carried out in an effective manner. I was then under the impression that the *volume* or *quantity* of alkalized urine which flowed over the stone was of great importance, and that the rate of dissolution would be proportionate to the rate of flow. Subsequent experiments have shown that this is not so, and that the *degree* of alkalescence is of much greater consequence. The large quantity of liquid in which the salt was dissolved diluted the urine unduly and greatly reduced its solvent power.

It is evident also that the dose of salt exhibited was too small. Rochelle salt, on account of its large quantity of crystallization-water, has less alkalizing power, by more than one third, than an equal weight of the citrate of potash; so that the 20-grain dose which the lad took in the first three weeks corresponded only to about 13 grains of the citrate, and the 30-grain dose administered in the second three weeks corresponded only to about 20 grains of the citrate.

Notwithstanding these drawbacks, it is not possible but that a considerable amount of dissolution had taken place. The urine was kept constantly, though feebly, alkaline for six weeks; there was no carbonate of ammonia developed in it, and no traces of phosphatic deposit on the stones. These are conditions in which, as the experiments already detailed inform us, uric acid cannot do otherwise than dissolve. The two calculi, when extracted, weighed only 22 grains, and yet one or both of them must have existed in the bladder for a period of three years. It is scarcely conceivable that in so long a time they had not attained a greater magnitude than this. And it seems not too much to suppose that had the treatment, imperfect though it was, been persevered in for another week or fortnight, the size of the concretions would have been sufficiently reduced to permit their escape spontaneously by the urethra.

The absence of granulations on the surface of the calculi,

with their polished, water-worn appearance, would have been highly indicative of solvent action had there been but one stone n the bladder; but the presence of two robs this character of its distinctiveness, inasmuch as a similar effect might have been produced by their mutual attrition.

Case 2.—J. C—, æt. 12, a healthy boy, was an inmate of the children's hospital in this town, under the care of Dr. Borchardt and Mr. Smart, who kindly permitted me to direct the treatment.

Symptoms of stone in the bladder had existed for a long time, and on sounding no difficulty was experienced in detecting a calculus. The urine was always found acid; it contained a moderate quantity of pus, and deposited crystals of uric acid freely on standing. No oxalate-of-lime crystals were detected at any time, nor, of course, any triple phosphate. The urine, in fine, was natural, except that it contained pus, and had an inordinate tendency to deposit uric acid. The bladder was somewhat irritable.

The stone was conjectured to be uric acid, but it might be composed of oxalate of lime or of a mixture of these two substances, which, indeed, it proved to be.

On the 19th of September, 1860, the lad was directed to take 20 grains of acetate of potash in two ounces of water every three hours. The urine was speedily rendered alkaline. The medicine was continued in the same doses with great regularity and without change for a period of thirty-four days. No particular alteration in the general state of the patient took place during this time. The appetite and sleep continued good, and the vesical symptoms were not more troublesome than usual. Frequent micturition was the chief inconvenience complained of. In the last ten days of the treatment a few crystals of triple phosphate were found in the urine after standing a while, but the secretion was perfectly sweet when voided, and no slimy mucus came away with it.

The stone being still found on sounding, after thirty-four days of treatment, operation was decided on, and the stone

was successfully extracted by Mr. Smart on the 24th of October. The patient recovered without a bad symptom.

The calculus measured one inch and three eighths in length, and an inch in breadth; it weighed 180 grains, and its form was a flattened oval.

It was found on examination to be composed of alternating layers of uric acid and oxalate of lime, and its surface presented a most peculiar appearance, which I shall endeavour to describe with the aid of the annexed illustration (Plate IV).

The outermost layer consists of yellow uric acid (figs. 1, 2, 3, *u*, *u*, *u*), and over the larger circumference of the stone it has a thickness in its deepest part of about a line and a half; but on the flattened surface this layer is dissolved away, and the subjacent layer of oxalate of lime crops through it to a considerable extent. On one side the patch of exposed oxalate (fig. 1, *o*) is as large as a sixpence, and presents the ordinary tuberculated surface and dark-brown colour of a mulberry calculus. On the opposite side two islets of oxalate are uncovered, each about the size of a large split pea (fig. 2, *o*, *o*). These are separated from each other by an intervening isthmus of uric acid.

Surrounding these exposed patches of oxalate of lime on the flattened surfaces are found the remains of a thinner, lighter-coloured, more superficial and incomplete layer, also composed of oxalate of lime. The irregular patches of this incomplete stratum occupy a higher level than the surrounding surface of uric acid (figs. 1 and 2, *o' o' o'*); and here and there little elevations of uric acid can be seen, surmounted with a shield of oxalate of lime (figs. 1 and 2, *e*). These elevations are partially undermined; the uric acid has been attacked by the solvent, and the protecting shield of oxalate of lime was in the process of being thrown off by the gradual melting of its support.[1]

[1] To compare small things with great, these capped elevations resembled both in appearance and their mode of origin the "glacier tables" of the Alps. Forbes, in describing them, says, "These consist of masses of rock lying on their flat sides, and supported above the general level of the glacier by an icy pedestal. This recalls our attention to a most important

The general surface of uric acid has a characteristic water-worn appearance, which is well represented in figs. 1 and 2. There are no minute mammillations such as usually beset the surface of uric-acid concretions; but the surface is undulating, and the hollows and intervening ridges are perfectly smooth. This undulation of the surface is evidently due to a want of uniformity in the composition of the stratum—the hollows corresponding to the spots where the uric acid is purest, and the ridges to the spots where it is mingled with a larger proportion of oxalate of lime.

The specimen is a remarkable one; it furnishes the clearest evidence yet adduced of the solvent action of alkalized urine or uric-acid calculi within the bladder.

No trace of phosphatic deposit existed on any portion of the stone.

Complete solution of the calculus was impossible in this case. A concretion composed of a uniform mixture of uric acid and oxalate of lime was found to be attacked with considerable facility by a solution of carbonate of potash in the phial; and the present specimen shows that thin and incomplete layers of oxalate of lime may be undermined and disintegrated by alkalized urine; but if the stratum of oxalate be complete and entirely invest the stone, it puts necessarily an absolute bar to further solvent action. This was the case in the instance before us. The partially uncovered layer of oxalate of lime surrounded the entire stone, and as soon as the dissolution of the superincumbent layer of uric acid had been completed no further diminution of size could have taken place.

The solvent treatment was inefficiently carried out in this

circumstance of glacier economy—that there is a perpetual waste at its surface. The action of the stone (mass of rock) is very evident; if the thickness of it be considerable, it forms a pretty complete shelter against the direct action of the sun's rays as well as against the contact of warm rains and wind." ('Tour of Mont Blanc,' p. xxiv.) In this comparison the uric acid corresponds to the ice, the oxalate of lime shields to the superincumbent mass of rock, and the alkaline solvent to the sun's rays and the warm rains and wind.

case. The dose of the acetate should have been nearly double. This would, probably, have more than doubled its solvent effect. The alkalescence of urine produced in a boy of twelve by twenty grains every three hours is only feeble, and does not approach the highest solvent power capable of being communicated to the urine.

Case 3.—Albert B—, æt. 6, was admitted into the Manchester Infirmary, under my care, on January 27th, 1862. On sounding, a calculus was found in the bladder. The patient was in good general health, and the vesical symptoms were quiescent.

The urine was repeatedly examined shortly after his admission; it was acid, clear, free from pus, blood, and albumen; it deposited uric acid freely on standing. No oxalate of lime or other unnatural ingredient was found in it.

From the absence of oxalate of lime and cystin, the calculus was conjectured—erroneously, as it turned out—to be composed of uric acid. A phosphatic stone was, of course, incompatible with a persistently acid state of the urine.

The patient was directed to take twenty grains of citrate of potash in six ounces of water every two hours. Three days subsequently, the dose was increased to twenty-five grains every two hours, in the same quantity of water. This quantity was administered regularly during the day, and moderately so during the night. The treatment was continued without change for a period of two months. The dose was then raised to thirty grains, every two hours, in eight ounces of water, and continued for a month longer, so that the entire period of treatment extended over three months.

The boy's health and spirits were excellent during this interval, and the vesical symptoms remained quiescent.

I am indebted to my former pupil, Dr. Gwyther, who was then my clinical clerk, for a most laborious and exact account of the state of the urine. The secretion was invariably alkaline. The degree of alkalescence was determined on eighty different occasions. The average of whole numbers

was 35 grains of carbonate of potash to the pint; sometimes it fell as low as 8 or 10 grains to the pint; sometimes it rose to 70, 90, and on one occasion to 120 grains to the pint. The average density of the urine was 1012, and the range extended from 1003 to 1022. The daily quantity, measured on three successive days, averaged 84 ounces.

Towards the latter period of treatment some pus-globules appeared in the urine; but the secretion was always sweet when voided, and quite free from any ammoniacal odour.

On the 7th of May the boy was sounded, and the stone easily detected. He was then transferred to the care of Mr. Southam, who extracted the stone by the lateral operation. With the exception of some degree of fever for a couple of days, the recovery was uninterrupted.

The stone was a typical specimen of the mulberry calculus. Its surface was exceedingly rough, and covered all over with spiny tubercles. Not a trace of phosphatic deposit existed on any part, and not the slightest evidence of solution could be detected. The stone was spherical in shape, and about as large as a good-sized marble. When sawn across, a nucleus composed of uric acid was displayed. Around this were some additional layers of the same substance. The crust of oxalate of lime was about a line and a quarter thick.

The solvent treatment was carried out in this case with undoubted efficiency for the space of three months. The only defects which my later experiments enable me to point out are the unnecessarily large amount of liquid taken, and the unnecessarily frequent repetition of the dose. If half the quantity of liquid had been administered an equal effect might have been produced with the same dose of the salt, repeated every third instead of every second hour.

These three observations permit a deduction of great importance to be drawn from them, namely, *that a continuously alkaline state of the urine does not determine any precipitation of the earthy phosphates, so long as the urine is free from ammoniacal decomposition.*

SECTION VIII.—*Discrimination of the cases in which the solvent treatment is, and is not, applicable.*

The first and most general limitation is—

The solvent treatment is inapplicable to all cases in which the urine is alkaline.

The loss of the acid reaction of the urine in calculous cases is due, in an overwhelming majority of cases, to ammoniacal decomposition of the urine in consequence of vesical catarrh.[1] This state of the urine determines the precipitation of a phosphatic crust on the surface of the stone, and withdraws it completely from the influence of alkaline solvents.

If the urine preserve its natural acidity the case may be regarded, *prima facie,* as suitable to the solvent treatment; but there are still numerous limitations which reduce the cases really suitable to a much narrower compass.

(*a*) In the first place, *all those cases are excluded in which it is known or strongly suspected that the stone is composed of oxalate of lime.* This is sometimes ascertained from the patient having previously voided concretions of oxalate of lime; sometimes the character of the urine indicates the nature of the stone. If it deposit on cooling an abundant sediment of octahedra or dumb-bells, the stone may be inferred, with great certainty, to be composed wholly or in part of oxalate of lime.

(*b*) When the examination of the urine and the previous history of the patient give *no indication of the nature of the stone* we are left in doubt (supposing the urine to be acid) whether it is composed of oxalate of lime, or uric acid, or of alternating layers of these two substances. There are no data at hand for an opinion as to the probabilities here involved. Different countries, and even different districts of the same country, show considerable diversities in the rela-

[1] The only exceptions are those very rare instances of calculi consisting of bone-earth or carbonate of lime. In these cases the urine is alkaline without being necessarily ammoniacal.

tive proportion of uric-acid and mulberry calculi. Calculi situated in the kidneys also differ essentially in regard to this point from vesical calculi. The former are generally composed of a single substance, and in about five sixths of the cases this is uric acid. The latter, if they have sojourned any considerable time in the bladder, are very frequently composed of two or more substances arranged in alternate layers. And here in reality lies the chief difficulty of the solvent treatment as applied to vesical calculi.

In cases of *renal calculi* the patient should evidently have the benefit of the doubt. No other treatment than that by alkaline solvents is open to the choice of the practitioner, and if the calculi should turn out to be composed of oxalate of lime the alkaline treatment will not aggravate, if it do not ameliorate, the state of the patient.

In cases of *vesical calculi* the question stands differently. The solvent treatment here comes into competition with the mechanical methods of lithotomy and lithotrity, which long experience has stamped with success. And this is not all, for it comes into competition in the very cases (small stones of uric acid or cystin with a healthy state of the urine) in which these methods attain their highest safety. It is no longer a question of the mere possibility of removing a calculus by means of solvents, but of doing it with less risk than by lithotomy or lithotrity.

Future experience alone can decide whether it is better in cases of this class (where the nature of the stone is quite uncertain) to consign them at once to the operating-table or to give a preliminary trial to the solvent treatment. It would appear from the cases reported in the preceding section that patients who have undergone such a trial may be afterwards transferred to the surgeon with undiminished chances of a successful operation. Probably the most advantageous course to follow, if the stone be a small one, would be to try the solvent treatment for a limited period—for six weeks or two months—and, if unsuccessful at the end of that time, to proceed without further delay to operation.

(*c*) *When the stone is known to be a large one* the solvent

treatment should not be attempted. The existence of a large stone in the bladder is itself an ever-present source of danger, and the larger it is, the greater the probability that it contains one or more layers of oxalate of lime which will resist the solvent. The length of time which a stone above the weight of an ounce would require for dissolution, also detracts greatly from the advantages of the solvent treatment, as compared with the swift though more hazardous method of lithotomy.

(*d*) The cases of vesical calculi *which are especially suitable to the solvent treatment are those in which it is known or strongly inferred that the concretion consists of uric acid and has not yet attained any great size.*

It sometimes happens than an individual voids spontaneously, from time to time, small calculi of uric acid, and that afterwards he becomes the subject of vesical calculus. Some one of the concretions, being perhaps larger than its predecessors, is unable, after its descent into the bladder, to pass out along the urethra as previous ones have done. After a season of rest the bladder begins to resent the presence of the foreign body, and symptoms of stone in the bladder awaken the patient to a knowledge of his situation. In such a case a dissolution of twenty or thirty grains from the surface of the stone would reduce its size sufficiently to enable it to traverse the urethra. This is what occurred in all probability in the person of Dr. Jurin, as related in a previous part of this paper. I am not unaware that in the case supposed, lithotrity, in the hands of a practised operator, who could in two or three sittings pulverize the stone effectually, subjects the patient to a minimum of risk, yet it is clearly a far greater risk than the use of acetate or citrate of potash for two or three weeks.

(*e*) It is probable that the solvent treatment, judiciously carried out, will form a useful *adjunct to lithotrity.* It is, however, essential to its employment than no vesical catarrh with ammoniacal decomposition of the urine, ensue after the operation. If the urine maintain its acidity after the stone is crushed, and if the fragments discharged prove to be uric acid, then the solvent treatment might be expected to act

advantageously by obviating the inconvenience and danger of repeated sittings.

To sum up in the affirmative—the solvent treatment is only applicable in those cases of vesical calculi in which *the urine is acid, the stone not large, its composition known to be uric acid (or cystin), or strongly suspected to be such.*

Section IX.—*Rules for carrying out the solvent treatment.*

(*a*) The action of alkalized urine is essentially slow; quick solution, by any manner of applying it, is impossible. To make up for this defect its operation must be *continuous and incessant*. To rest content with alkalizing the urine for a few hours each day, is not only to reduce the solvent effect to an insignificant quantity, but sometimes, at least, to nullify it altogether. I have known urine, kept continuously alkaline by acetate of potash for several successive days, recover its acidity and deposit uric acid within a few hours of the latest dose. So that it is possible, with an insufficient maintenance of the alkalescence of the urine, that the dissolution effected during one part of the day may be nullified by fresh deposition of uric acid during another part of the day. It is also of the utmost importance not only to keep the urine continuously alkaline, but to *keep it alkaline to a certain degree.* The experiments described in the second section of this paper prove that solutions with an alkalescence below three grains of carbonate of potash to the pint, have scarcely a greater effect on uric acid than common water. A feebly alkalized urine acts so slowly that, in cases of vesical calculi, the delay incurred, counterbalances the safety of the treatment as compared with mechanical means, and robs it of the preference which it might otherwise deserve.

To secure a continuous alkalescence, the dose must be repeated at short intervals during the day, and, as far as possible, during the night. An interval of two or three hours between each dose is sufficiently short to ensure an adequate constancy of effect, and is not burdensome in practice.

During the waking hours perfect regularity can be easily maintained. A dose should be taken the last thing before retiring to rest. This will probably necessitate the emptying of the bladder once at least in the course of the night, and furnish an opportunity to take another dose, which will carry the patient to the time of waking. Indeed, patients with vesical calculi are rarely able (quite apart from the effect of medicine) to pass the night without awaking once or more to relieve the bladder.

(*b*) The most convenient salts for alkalizing the urine are the citrate, acetate, and bicarbonate of potash. The percentage of base in each is almost the same, so that they may be given in equal doses. The acetate and citrate of potash seem, on the whole, to deserve the preference. The citrate, as found in the shops, is generally impure, and contains a good deal of insoluble matter. It is better to make a solution of fixed strength from the crystallized bicarbonate of potash and crystallized citric acid. The following prescription yields a solution containing a drachm of the citrate in each fluid ounce:

℞ Potass. bicarb., ʒxij;
Acidi citrici, ʒviij + gr. xxiv;
Aquæ ad ℥xij. Solve.

A fluid ounce of this solution mixed with three or four ounces of water, makes a draught which has scarcely any taste, and which even children take without any difficulty.

For an adult, the dose should not be less than 40 or 60 grains, and for children not less than 20 to 30 grains, every third hour, dissolved in three or four ounces of water. If the dose be given in less water than this, the patient is apt to be a little thirsty and to drink capriciously in the course of the day, thereby introducing an unnecessary irregularity into the flow of the urine and an excessive oscillation in the degree of alkalescence.

(*c*) It is essential that the freshly voided urine should be frequently examined during the progress of the treatment. If at any time it show signs of ammoniacal decomposition

the treatment should be given up. The advent of this state is recognised by the offensive ammoniacal smell of the secretion, the increase of pus and flaky matter in it, and the speedy gelatinization of the pus into a viscid mass. As long as the urine continues *sweet when voided* no fear need be entertained of the deposition of the mixed phosphates on the surface of the stone.[1]

SECTION X.—*An examination of some of the objections which have been urged against the principle of the solvent treatment.*

(*a*) The most plausible objection against the alkaline treatment is the alleged danger of the precipitation of the phosphates on the surface of the stone. The facts advanced in the preceding pages dispose of this objection completely. If there be ammoniacal decomposition of the urine, the phosphates are deposited whether alkaline medicines be given or not, and the concretion goes on increasing; but if the urine be alkaline solely from fixed alkali not a particle of phosphatic deposit takes place.[2]

(*b*) It has been said that the natural reaction of the urine is acid, and, therefore, that rendering it alkaline introduces an unnatural state, which cannot fail to act deleteriously on the general health. In a state of fasting the natural healthy urine is doubtless always acid, but the researches of Dr. Bence Jones, fully confirmed by my own, show that the urine is normally alkaline (from fixed alkali) for several

[1] Means might be easily suggested for restoring the urine to its natural state after it has become ammoniacal from vesical catarrh, with a view to the resumption of the solvent treatment; but it is pretty evident, at least in the present position of the question, that a solvent treatment so complicated could not compete with the mechanical methods.

[2] A want of knowledge of the essential difference of properties between urine alkaline from fixed alkali and urine alkaline from carbonate of ammonia, runs like a thread of error through all the elaborate argument of Civiale, in his chapter on the dissolution of the stone in the interior of the body. (See chap. iv of his work 'Du traitement médical de la Pierre.')

hours daily, after meals, in many, if not in all, healthy persons. So that the maintenance of an alkaline reaction of the urine by fixed alkali is by no means so unnatural a state as some have supposed.

(*c*) Alkaline substances, it is urged, impair digestion. This objection was valid against the ruder methods of alkalizing the urine formerly employed. But the acetates and citrates have no such effect. The introduction of these salts, in recent times, for the treatment of articular rheumatism, has afforded an immense field for watching their effects. Indeed, the solvent treatment here recommended is identical with the prevailing mode of treating rheumatism, except that the dose is administered in a somewhat more dilute form. In the last eight years I have employed the bicarbonate, the acetate, and citrate of potash, both in private and public practice, in quantities of four, six, and eight drachms in the twenty-four hours, in a very large number of cases. The majority were cases of articular rheumatism, the remainder embraced a variety of slighter and more severe disorders—skin diseases, emphysema, diabetes, acute Bright's disease, &c. The urine was kept continuously alkaline for periods varying from a fortnight to three months, and in no instance were deleterious effects observed. In a patient with emphysema of the lungs the urine was kept uninterruptedly alkaline for fourteen weeks, with marked improvement of the general health and steady increase of weight. In short, the acetate and citrate of potash have appeared to me about as harmless as so much sugar.

Appendix to Part I.

Experiments having reference to the solvent treatment of cystin calculi.

Cystin is soluble both in the carbonates of the fixed alkalies, and in mineral acids. It may therefore be attacked, when existing as a calculus in the bladder, either by alka-

lizing the urine, as in the solvent treatment of uric acid, or by injecting acid solutions into the bladder.

Two experiments were performed with a view of testing the solubility of cystin calculi in a solution of carbonate of potash containing 40 grains to the pint.

A section of a pure cystin calculus, weighing 97 grains, was employed. In the first experiment two pints of the solution were passed over the fragment, at blood heat, in the course of twelve hours. The loss of weight, at the end of that time, amounted to 9⅛ grains.

In the second experiment, similarly performed, but with a flow of three pints, the loss of weight, in twelve hours, was 10½ grains.

These experiments yield a rate of solution of about 20 per cent. in twenty-four hours. Cystin may, therefore, be regarded as being even more favorable to the application of the alkaline solvent treatment than uric acid.

PART II.

SECTION I.—*Experiments on the solvent treatment of uric-acid calculi by injections into the bladder.*

It has been conceived that considerable advantage would be gained in cases of vesical calculi if the solvent were directly injected into the bladder by means of a double-current catheter, instead of being made to pass by the more circuitous route of the circulation and the secreting tissue of the kidneys.

The advantages which the injection method seemed to offer were—

(*a*) A much greater mass of the solvent could be passed over the surface of the stone in a given time.

(*b*) The solvent could be used in a more concentrated form.

(*c*) Certain substances could be used as solvents which are not capable of reaching the stone through the medium of the circulation.

(*d*) The inconvenience of a long course of medicines in large doses would be avoided.

The following experiments, however, show pretty conclusively that with respect to uric acid these advantages are illusory.

The mode of proceeding adopted was, to place a section of a uric-acid stone in a ten-ounce phial, and to pass over it at blood heat a current of the solvent to be tried, as large as the capacity of the urethra might be supposed to permit.[1] The current was kept up for two or three hours continuously.

At first, saturated solutions of bicarbonate of potash and the common phosphate of soda were employed, but they failed to make the least impression on the stone. Afterwards solutions containing 240 grains to the pint were used, with a like result. I was not then aware that the maximum solvent power of these, and kindred salts, on uric acid, lay in much weaker solutions. After attaining to this knowledge I employed a solution of *carbonate of potash* containing 50 grains to the pint, which represents the maximum solvent power of that salt on uric acid.

Two experiments, each of three hours' duration, yielded the following results:

Hourly rate of flow of the solution . .	42 pints.
Weight of the fragment of uric acid . .	57 grains.
Hourly dissolution	2 „

This result, insignificant as it is, could probably not be approached in the living bladder, on account of the mechanical difficulties to be overcome.

Four experiments with *carbonate of lithia* yielded still smaller results.

[1] The method of treating vesical calculi by injections was proposed so long ago as 1744 by the Rev. Dr. Stephen Hales, who invented the double-current catheter for the purpose; it was further investigated by Langrish in a large number of experiments on bitches. 'Physical Experiments on Brutes,' by Browne Langrish, London, 1746.

A solution of 10 grains to the pint, with an hourly flow of thirty pints, dissolved less than a grain per hour.

A solution of 20 grains to the pint, with an hourly flow of twenty-six pints, dissolved one grain and a quarter per hour.

Solutions of the following substances were also tried in a similar manner, namely, *borax, borax with liquor sodæ, double borate of potash and soda, common phosphate of soda, basic phosphate of soda,* and *potash soap,* but their solvent effects never reached beyond a loss of weight (on a fragment of uric acid weighing about 100 grains) of one or two grains in the hour.

Lime water, in a continuous current, at the rate of thirty pints an hour, dissolved a fragment weighing 86 grains, at the speed of one and a half grain each hour.

Seeing the very small results thus obtained, I proceeded to try the caustic alkalies, which are the most powerful known solvents for uric acid.

Sixty minims of the *liquor potassæ* of the London Pharmacopœia (sp. gr. 1063) were added to each pint of water. A solution so made was perceptibly acrid in the mouth. Fifty-six pints of this solution dissolved in an hour two grains from a fragment weighing 100 grains.

Twenty pints of a solution containing 120 minims of liquor potassæ to the pint dissolved also two grains per hour.

Twenty-four pints of a solution containing 240 minims to the pint dissolved nearly three grains per hour.

Liquor sodæ (sp. gr. 1056), in the proportion of 120 minims to the pint, dissolved two grains per hour with an hourly current of twenty pints.

A solution of the same, containing 240 minims to the pint, with an hourly current of thirty-four pints, dissolved two and three quarter grains per hour.

The stronger solutions of the caustic alkalies had so great a pungency in the month, that it seemed impossible that they could be safely used as injections into the bladder.

The general conclusion from all the foregoing experiments

is, that under the most favorable conditions, and with the most effective solvents capable of being borne by the living bladder, no greater dissolution than one or two grains per hour can be accomplished in the case of uric-acid concretions. In actual practice the conditions would necessarily be much less favorable than in an experiment performed in the laboratory. A little consideration is sufficient to show that these results hold out no prospect of any useful practical application.

Section II.—*Experiments on the solvent treatment of oxalate-of-lime calculi.*

It has been asserted by Chevallier[1] and Ch. Petit[2] that the alkaline carbonates produce slow solution of oxalate-of-lime concretions. This assertion is based on experiments made by immersing mulberry calculi in the warm springs of Vichy. The enormous quantity of the solvent flowing over the calculi, under these circumstances, involves, however, a condition which is unattainable in the living body, and no practical inference can therefore be drawn from such observations.

I found that a flow of six or eight pints per twenty-four hours of a solution of *carbonate of potash* containing 40 grains to the pint had no perceptible effect on a calculus of oxalate of lime.

In the case of Albert B—, reported in the first part of this paper, alkalized urine flowed over a mulberry calculus for a period of three months, without producing the least sign of solution.

Better effects, it was conceived, might be obtained by a solution of *dilute nitric acid* (which is the best solvent of oxalate of lime), employed in a manner so as to imitate injections into the bladder.

A solution, containing 120 minims of concentrated nitric acid to the pint, was prepared; it was found to possess an

[1] 'Med. Gaz.,' vol. xx, p. 581.

[2] 'Mode d'action des Eaux minérales de Vichy,' p. 199.

acridity probably a good deal beyond what the living bladder could withstand; and yet twenty-four pints of this solution, passed in the course of an hour, at blood heat, over a mulberry calculus weighing fifty-three grains, only dissolved half a grain. The excessive hardness of oxalate-of-lime calculi accounts for this extreme slowness of solution.

There is little difficulty in concluding from these experiments that oxalate-of-lime concretions are practically unassailable by solvents applied in any known method.

Section III.—*Experiments on the solvent treatment of the mixed phosphates or fusible calculus.*

The observations of Chevallier and Petit on the effects of immersing phosphatic calculi in the Vichy springs led them to believe that rapid disintegration of these concretions would take place in solutions of the alkaline carbonates. The conditions of these experiments, however, bear no analogy to the actual circumstances of a phosphatic calculus in the bladder. Such a calculus is bathed in an ammoniacal urine, and the calculous matter continues to be deposited just as before, notwithstanding the addition of a fixed alkaline carbonate to the urine. An observation recorded in the first part of this paper, Section VI, places this matter beyond doubt.

Some direct experiments on the effect of solutions of *carbonate of potash* on phosphatic calculi were also made. Solutions containing 30, 40, and 80 grains to the pint were passed, at blood heat, at the rate of six pints in the twenty-four hours, over a rough, porous, phosphatic stone weighing 371 grains.

In two experiments there was no loss of weight; in three others a few fragments chipped off, and occasioned a diminution of weight amounting to two grains in the twenty-four hours.

The results indicated very clearly that alkalized urine could have no effect in the human bladder in dissolving

or disintegrating a calculus composed of the mixed phosphates.

Far more promising results were obtained by dilute nitric acid, used so as to imitate injections. The following experiment is to be regarded merely as rudely tentative.

A solution containing a drachm of the commercial nitric acid per pint was passed, at blood heat, over a phosphatic stone weighing 153 grains, at the rate of thirty-six pints in the hour. The loss of weight which followed amounted to twenty-one grains per hour. A modification of this proceeding was successfully employed, as is well known, by Sir B. Brodie, in actual practice. My colleague Mr. Southam has recently tried the same method, and with complete success. The stone had been repeatedly broken with the lithotrite, but new phosphatic concretions formed in the bladder as fast as the old ones were crushed, and it was found impossible to completely clear the bladder. In this difficulty an injection, containing two drachms of dilute nitric acid to a pint of water, was practised every day or every second day. In the course of a short time the old fragments were entirely dissolved, and the formation of new ones was prevented. The urine ceased to be ammoniacal, and the bladder was speedily cleared. This method seems capable of wider application than is now made of it by surgeons.

The following conclusions may be drawn from the experiments described in Part II:

1. That uric-acid calculi cannot be successfully treated by alkaline solvents used as injections into the bladder.

2. That oxalate-of-lime calculi are practically insusceptible to acid and alkaline solvents.

3. That phosphatic calculi are insusceptible to the action of alkaline solvents, but that they offer an encouraging prospect for the use of acid injections into the bladder.

DESCRIPTION OF PLATE III.

(See page 117.)

FIG. 1.—Upper flattened surface of the stone.

u u. Water-worn layer of uric acid.
o. Exposed patch of oxalate of lime.
o'o'. An imperfect, more superficial, partly undermined, lighter-coloured layer of oxalate of lime.

FIG. 2.—Lower flattened surface of the stone (the *letters* refer to the same parts as in Fig. 1).

FIG. 3.—Section of the stone vertically through the exposed patches of oxalate of lime.

Fig. 1.

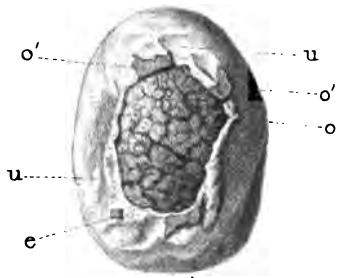


Fig. 2.

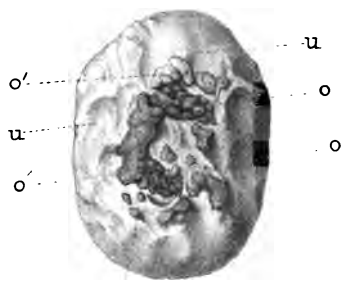
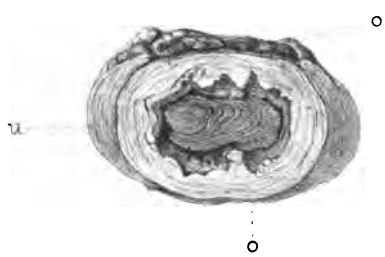


Fig. 3.



ON

DELIRIUM OR ACUTE INSANITY

DURING THE

DECLINE OF ACUTE DISEASES, ESPECIALLY THE DELIRIUM OF COLLAPSE.

BY

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THE mental aberrations occurring during the increase and height of acute diseases are among the most interesting phenomena of pathology, and it is probably only the frequency of their occurrence which prevents us from paying due attention to their nature and details; but I do not wish at present to direct the attention of the Society to this kind of delirium—the *delirium febrile*—but to the delirium or the acute insanity which occasionally breaks out when the disease has already entered into the stage of decrease, when the fever has almost or entirely ceased, and when, perhaps, the patient has just been declared convalescent—a delirium, therefore, which might be called the *delirium decrementi* (delirium of the stage of decrease), or, for reasons afterwards to be mentioned, the *delirium of collapse*.

I couple the term "acute insanity" with that of "delirium" because the nature of the mental aberrations, and the condition of the patient affected with it, are not the same as in the common febrile delirium, but resemble much more those which accompany the aberrations usually comprised under the term "insanity," and especially those described under the head of mania. I feel, however, that there does not exist a distinct line of demarcation between delirium and insanity.

The mental aberrations in question are not unknown in our literature; there exist, on the contrary, two valuable papers on the subject by Thore;[1] Griesinger has a very interesting paragraph on it, in his excellent work on mental diseases,[2] and other authors, too, have mentioned it in treating of various acute diseases, especially Chomel,[3] Louis,[4] Sauvet,[5] Trousseau,[6] and Tüngel,[7] in connection with typhoid fever, and Graves as one of the sequelæ of typhus (maculated typhus fever).[8] In spite of so long a list of names, however, which might probably be enlarged,[9] I have reason to conclude,

[1] "Etudes sur les maladies incidentes des aliénés." Par M. le docteur Thore. 'Annales Médico-psycholog.,' tom. iv, pp. 339—362. "De la folie consécutive aux maladies aigues." Par M. le docteur Thore fils. 'Annales Médico-psycholog.,' 1850, vol. ii, p. 586.

[2] 'Die Pathologie und Therapie der psychischen Krankheiten.' By Dr. W. Griesinger. Second edition, 1861, pp. 186 ss.

[3] 'Leçons de clinique médicale.' Par le professeur A. F. Chomel. Recueillis par J. L. Gouest, D.M.P. Paris, 1834, vol. i, p. 54.

[4] 'Recherches ... sur la fièvre typhoide.' Par P. C. A. Louis. Second edition, vol. i, p. 33, 1841.

[5] "Remarques sur le délire consécutif aux fièvres typhoides." Par M. Sauvet. 'Annales Médico-psychol.,' vol. vi, p. 223, 1845.

[6] 'Clinique Médicale de l'Hôtel-Dieu de Paris.' Par A. Trousseau. Vol. i, p. 189, Paris, 1861.

[7] 'Klinische Mittheilungen von der medic. Abtheilung des allgemeinen Krankenhauses in Hamburg.' Von Dr. C. Tüngel. Hamburg, 1859—1864.

[8] 'Clinical Lectures on the Practice of Medicine.' By Robert J. Graves, M.D. Second edition. Edited by J. Moore Neligan, M.D. Vol. i, pp. 256 ss, Dublin, 1858.

[9] Since this paper has been transmitted to the Society Dr. Gairdner, of Glasgow, has published a clinical lecture on typhus fever, in which he likewise alludes to a delirium showing itself for the first time after the patient

from frequent conversations on the subject, that many medical men have never observed this kind of delirium; and the fact of my having repeatedly met with it in a comparatively small number of years is perhaps owing only to that peculiar caprice, well known to most of us, whereby it happens that remarkable and rare forms of disease occur sometimes in clusters to one man in a certain period, while they are but rarely or never seen by others, or by the same observer at other periods.

I will give the outlines of the cases which I have observed before I venture any further remarks.

The first case occurred in the Policlinical Institution at Bonn, while I was assistant-physician to the late Professor F. Nasse.

Case I.—A. L—, an apparently healthy lad, æt. 16, had an attack of *measles* with broncho-pneumonia in January, 1851; the eruption, which was very general, had been preceded during five days by the well-known cough; the fever reached its climax on the seventh day of the disease, or the second day of the eruption, and decreased considerably on the eighth day; the broncho-pneumonia, too, was in a state of resolution, and the patient began to enjoy light food. On the ninth day, immediately after waking, the lad, who never had been delirious during the active stages of the disease, began to cry, saying that his employer had dismissed him on account of suspected dishonesty, and that he was to be taken to prison; he constantly asserted that policemen were at the door and he endeavoured to jump out of the window; he shouted to imaginary people whom he fancied to hear calling him from the street, and had occasionally also hallucinations of sight, mistaking friends who were present for people connected with his delusion; it was useless to tell him that he had been in bed for more than a week, and had not seen his employer since then; he listened for a moment, but returned immediately afterwards

has begun already to show symptom of crisis. "On the Course of Typhus Fever, and especially on the Phenomena of Crisis." By W. T. Gairdner, M.D. 'Lancet,' 1865, vol. i, p. 59.

to his delusion. The pulse was under 100, very feeble; the temperature not increased, the skin bathed in perspiration, the eye anxious; but he ate and drank with some appetite. The whole aspect of the lad was not that of the delirium of fever, but that of a kind of mania. The treatment consisted in the administration of half a grain of opium every three hours.

During the night he had a few hours' sleep, with loud dreaming. On the following day he was not yet clear, but had the idea that his father was in prison for some political offence; he was, however, calmer than on the ninth day; pulse still feeble; temperature normal; he perspired freely; passed a large quantity of urine (free from albumen), and had a good motion. Instead of half a grain of opium every three hours, a quarter of a grain of morphia was given every four hours. He had rather more sleep than during the preceding night, and on the eleventh day was almost rational, but had a vague expression of face, and was still inclined to believe in his father's imprisonment, until he was, by the appearance of the latter, assured of the contrary. After nine hours' sleep in the night from the eleventh to the twelfth day the patient awoke perfectly calm and clear, ascribing all his delusions to vivid dreams. From that time he made a very good recovery. He had never been delirious either during or after any previous illness.

Some medical men who had seen the patient with me were inclined to regard his mental derangement as caused by metastasis of the eruption to the brain; but there was not the slightest show of reason for this view, as the eruption had not faded any sooner than it does in most other cases.

In connection with measles I observed another case, which is heightened in interest by the facts that there were two distinct outbreaks of delirium, and that the delirium occurred besides at a later period in the same patient during the decline of scarlet fever.

Case II.—M. M—, æt. 13, a very excitable, otherwise healthy girl, after having had a peculiar harsh cough for eleven

days, with slight pyrexia, manifested the eruption of *measles* on March 22nd, 1857. Pyrexia moderate; at 4 p.m., temperature 39° cent. (102°2 Fah.); pulse 110. (Effervescing tartrate of soda and potash.) On March 23rd the eruption was well out; pyrexia moderate during the morning, but towards the evening vomiting, headache, great excitement, slight delirium; at 5 p.m. pulse 136, irregular; temperature 40°8 cent. (105°44 Fah.). (Tinct. Opii ɱx.)

March 24th.—Night better; no more delirium, no sickness; eruption very general; mind free and quite calm; at 4 p.m. pulse 124; temperature 39°5 cent. (103°1 Fah.).

25th, 5 p.m.—Eruption slightly paler; no delirium whatever; pulse 116; temperature 39°2 cent. (102°56 Fah.).

26th, 10 a.m.—Eruption still quite distinct, but paler; scarcely any pyrexia; pulse 98, very feeble; temperature 37°2 cent. (98°96 Fah.); mind calm; every symptom favorable. Sudden outbreak of violent delirium on the same day in the afternoon. When I saw her in the evening she was very excited, and could scarcely be restrained in the room, asserting that there was fire in the poultry yard; that she must save the chickens, else they would all be burnt. The surface of the body was by no means hot, the temperature in the axilla being exactly the same as in the morning; pulse 95; impulse of heart weak; profuse respiration; expression of great anxiety. (A quarter of a grain of acetate of morphia every three hours.)

27th.—Has become calmer after midnight, and slept soundly in the morning; was rational on waking, but could be persuaded only with difficulty that there had been no fire. Takes light food; bowels regular. Urine passed in large quantity, with slight sediment of lithates, free from albumen and sugar.

At 4 p.m.—Pulse 75, feeble, rather irregular; temperature 36°8 cent. (98°24 Fah.).

(Morphia discontinued since the morning.)

28th.—Had felt so much better at noon that she went for a short time into another room. The pyrexia had entirely

ceased, the temperature being only 36°2 cent. (97°6 Fah.); pulse 90, weak. In the afternoon she was suddenly seized with headache, vomiting, and violent delirium, imagining that a great misfortune had happened to a near friend of hers; had hallucinations of hearing and also of sight. (Received at once a dose of morphia.) At 10 p.m., when I saw her, she was still excited, and occupied with her delusion, but calmer than in the afternoon; she was bathed in perspiration, and the skin felt cool to the hand, the temperature in the axilla being 36°4 cent. (97°52 Fah.); the pulse 94, rather irregular. Towards midnight she fell asleep, after having taken three doses of morphia.

On the 29th, on waking, she was quite calm and rational, but could at first scarcely believe that her fear had been unfounded.

After this the convalescence progressed very favorably. The nature of the delusions could be distinctly traced to some stories which she had read with great interest just before her illness.

She enjoyed good health up to the middle of December, 1858, when she had a severe attack of *scarlet fever*. The eruption appeared on December 16th, and was very general; the suffering from the throat and tongue was great, and the fever unusually high, the temperature reaching on the 18th, at 5 p.m., 41°4 cent. (106°52 Fah.), with a pulse of 155 to 160, and great depression of power; yet the patient was never delirious while her eyes were open, and but slightly so during sleep. A great abatement of all the morbid symptoms took place on December 20th, when the temperature decreased between the morning and evening from 39°4 to 38°5 cent. (102°92 to 101°2 Fah.), the pulse being 118 in the morning and 116 in the evening; the urine being copious, forming a a thick sediment of lithates, and containing but slight traces of albumen.

On December 21st the improvement of the general symptoms continued; the soreness and the swelling of the throat and tongue, however, were still considerable, preventing her from taking sufficient support. The temperature at 4 p.m.

was 38°25 cent. (100°85 Fah.); the pulse 115, rather feeble. The eruption had become much paler.

On the 22nd, at 4 p.m., temperature 38°2 cent. (100°76 Fah.); pulse 112; patient took rather more nourishment; urine free from albumen.

On the 23rd, in the evening, when her pulse had been at 7 p.m. 116 and feeble, the temperature only 37°8 cent. (100°04 Fah.), she suddenly became delirious in waking from a short sleep. The delirium was of an anxious character, about the death of a near friend and the severe illness of another. She asserted that she had seen a mutual friend who had brought the news direct from the death-bed of the one and the sick-bed of the other; she had frequently hallucinations of hearing, cried almost constantly, and could with difficulty be kept in bed. She was in profuse perspiration; hands and feet were cold. After two doses of one third of a grain of acetate of morphia, the delirium having lasted scarcely eight hours, the patient fell asleep, and slept with little interruption eight hours.

On waking on the 24th she was quite rational, but it required the serious and repeated assurance of all her friends to convince her of the unreality of her delusions. The pulse at 2 p.m. was 105, the temperature 37° cent. (98°6 Fah.), the skin uniformly warm, and moderately moist. "It must have been a dream," she told me, "but a very dreadful one." Again the subject of the delirium seemed to be connected with a story read shortly before the commencement of the scarlet fever.

She made an excellent recovery from that time, and has never since had the slightest derangement of her mental faculties.

I may here mention that the late Dr. Addison, who had repeatedly seen the patient with me, often said at the time and afterwards that it was perhaps the most severe case of scarlet fever he had ever seen recover. This distinguished physician ascribed the peculiar delirium to the scarlet-fever poison, and brought it in connection with the intensity of the symptoms during the former stage of the disease. Often as he had met with delirium during the increase and height of scarlet

fever, and even at the very commencement of the disease, before the outbreak of the eruption, he never, he said, had witnessed such an outbreak during what might be called the convalescence.

A not less remarkable instance occurred in connection with *erysipelas of the face and head* in a female servant, who had within four weeks two distinct attacks of erysipelas, separated by a perfectly free interval, each of these attacks being attended at the eve of convalescence with temporary insanity. The patient was an inmate of the German Hospital, where I had the advantage of attending her as resident-physician, with Dr. Swaine, of York, who at that time was one of the physicians of the hospital.

Case III.—M. E—, æt. 29, an otherwise healthy female servant, of rather excitable disposition, was admitted into the German Hospital on January 3rd, 1852, with erysipelas of the left side of the face and forehead. She had been ill five days; the erysipelas was well out, forming large blisters on the cheeks and forehead; the fever was moderately high, by no means excessive; the temperature varying on the 3rd, 4th, and 5th, between 38°8 and 39°8 cent. (101°84 and 103°64 Fah.); the pulse between 104 and 112, delirium being altogether absent, and the other constitutional symptoms mild.

January 6th.—The local affection was subsiding; the patient felt in every respect better, and took some food; urine slightly acid, without albumen, sp. gr. 1024; temperature at 8 a.m. 37°8 cent. (100°04 Fah.); pulse 95; at 5 p.m. temperature 37°5 cent. (99°5 Fah.); pulse 88. The medicinal treatment, which had consisted of small doses of sesquicarbonate of ammonia, was discontinued.

7th.—Early in the morning the patient was found dressing herself, stating in great excitement that she must go to see her brother who was in prison at Rastadt,[1] and was shortly to be executed for fighting against his government in the in-

[1] Rastadt is a town and fortress in the Grand-Duchy of Baden, where in 1849 the insurrection broke out and terminated, and some of the insurgents remained as prisoners for some time.

surrection. She had no idea of her being in England, believed herself to be at Baden-Baden (where she had been in service four months ago, before she came to England); could not be persuaded that she was the inmate of an hospital, and could only with great difficulty be restrained in bed. Expression anxious, face pale and rather cold, as also hands and feet; profuse perspiration; temperature in axilla 37°2 cent. (98°96 Fah.); pulse 90, weak and irregular. The erysipelatous affection had further subsided; desquamation commenced.

To have one third of a grain of morphia every three hours. Nourishment and port wine in moderate quantity.

At 9 a.m. she was even more excited; she asserted that she had seen her father, who had told her she must go to her brother at once, as the execution was to take place soon. The expression was that of extreme anxiety; the face pale, the eyes sunken, nose and forehead quite cold, profuse perspiration on the head; hands and feet still cold. (Hot mustard poultices to arms and legs.)

At 6 p.m., after four doses of morphia, she was rather more quiet, but had still the same delusion; pulse 88 to 92, less feeble; temperature 37°3 cent. (99°14 Fah.). Face was pale and cold, as also hands and feet.

At 10 p.m., two hours after the fifth dose of morphia, the patient fell asleep, and slept with little interruption nine hours.

8th.—At 9 a.m. she was much depressed and felt very weak; she still believed, on waking, that she was in her native country, but after some conversation was persuaded of her error, and seemed then much relieved. Pulse 82, weak; temperature 36°8 cent. (98°24 Fah.).

9th, 10th, and 11th.—She felt well, and was out of bed, the temperature varying between 36°2 and 36°8 cent. (97°16 and 97°88 Fah.), but on the evening of the 11th the temperature had increased to 37°4 cent. (99°32 Fah.); on the evening of the 12th she had well-marked rigor, with a temperature of 38°6 cent. (101°48 Fah.), and a pulse of 108.

14th.—Erysipelas appeared on the right side of the forehead, spreading thence to the scalp. Urine free from albumen. No delirium. No unusual symptoms. The pyrexia declined on January 18th, and so, at the same time, did the erysipelatous affection. The patient felt herself better, was hopeful, and had commenced taking solid food, when on January 19th, at 3 a.m., she became again excited, and was found shortly afterwards, almost undressed, in the cold passage, endeavouring to leave the house, stating that her father had suddenly died and that she must join her mother, who had just sent her an urgent message. It was useless to argue with her; she again believed herself at Baden-Baden, and could not be convinced of the contrary. She bore, at the same time, the expression of great collapse of vital powers; face, hands, and feet cold; profusely perspiring; pulse 98, extremely weak and irregular; impulse of heart feeble.

Treatment.—One third of a grain of morphia every three hours; hot wine; hot poultices to hands and feet.

At 8 a.m., after having taken two doses of morphia, she was still quite as excited and deranged as before, but took her breakfast well; the symptoms of collapse were diminished; the face, the hands and feet, were less cold; pulse 102, rather stronger and more regular; temperature in axilla 37°2 cent. (98°96 Fah.). After five doses of morphia, and fourteen hours from the commencement of the attack, she fell asleep, and slept about seven hours. On waking the delusion had not yet left her; she wanted to dress herself at midnight; but a short time after another dose of morphia she fell asleep again, and slept till 8 a.m. (January 20th), when she was much astonished to find herself in the hospital, and gradually began to believe that she had again been under a delusion. She was comfortably warm, and took breakfast with much appetite. The pulse at 9 a.m. was 90, stronger and regular; the temperature 36°8 cent. (98°24 Fah.), but descended in the course of the following day to 36°2 cent. (97°16 Fah.). The further progress to recovery was uninterrupted.

With regard to the subjects of the delusions, it was after-

wards ascertained that a brother of the patient had really been in the Badish revolutionary army, but had never been sentenced to death; it was further found that she had received, during the interval between the two attacks of erysipelas, a letter from her relatives mentioning her mother's illness, but not her father's death.

In connection with *pneumonia*, I have met with two cases of this transitory mental derangement.

Case IV.—J. V. B—, æt. 64, a rather delicate and excitable, but otherwise healthy man, of temperate and industrious habits, came in December, 1861, from the Continent to London, where he had to transact important business.

After having had a slight cough for some time he was seized with rigor on December 15th, and had all the symptoms of right-sided pneumonia of moderate extent; the constitutional symptoms were in proportion to the local affection, the temperature never exceeding 39°5 cent. (103°10 Fah.), the pulse, however, being always rather frequent (between 100 and 130) and feeble; the patient's mind was anxious about the final result of the disease, and also about his inability to attend to business, but there was no delirium. The treatment consisted in rest, light nourishment, a moderate quantity of wine, and very small doses of nitrate of potash, with vinum stibiatum and laudanum.

On December 21st, at 6 p.m., the local affection was in full resolution; the appetite good; the patient himself more cheerful; the pulse had decreased to 88; the skin felt comfortably warm, slightly perspiring; temperature 37°2 cent. (98°96 Fah.). To Dr. Gueneau de Mussy, who kindly assisted me with his valuable advice, and also to myself, the patient appeared convalescent or on the eve of convalescence. However, between 1 and 2 o'clock in the following night, I was urgently summoned, and found the patient almost undressed on the stairs of the hotel, pushing away his wife and a female attendant, saying that he wanted to see a priest, as he was going to die in a few hours. I learned that he had been

in this state since soon after midnight, seeing strange people in the room, thinking them enemies and demons, endeavouring to strike them, hearing people talk about him in the street, having the conviction that he must die, and asking constantly for the priest to administer to him the sacraments of the Roman Catholic Church. The excitement seemed increased by the idea that his wife and those around him endeavoured to prevent his seeing his confessor, whom he believed to have arrived at the hotel. Coupled with this mental derangement was a great collapse of vital powers; face much altered and pale, eyes sunk in, nose sharp and cold, in fact, a *facies Hippocratica;* hands and feet likewise cold; pulse 140, scarcely perceptible.

Treatment.—One third of a grain of morphia with a drachm of ether every two hours; hot wine every half hour; warm poultices to hands and feet. At 8 a.m. he began to sleep, after having taken two thirds of a grain of morphia and two drachms of ether; slept much during the day, was quite rational in the evening, and believed his delusion to have been a terrible dream. The skin was warm, but he was free from fever, the temperature being 36°5 cent. (97°7 Fah.); pulse 88. The convalescence from this time was very satisfactory.

With regard to the causation of this attack of maniacal delirium, it may be stated that the patient's wife had arrived on the day before the occurrence, when he conversed with her much about his business, and that he had taken, in addition, a rather indigestible supper (lobster-salad) on the evening preceding the outbreak of the delirium.

Case V.—C. F—, æt. 22, of anxious disposition, but generally in the enjoyment of good health, came to London in the course of last summer to seek employment in a mercantile house. For some months he was unsuccessful, and when he had found a situation he perceived his knowledge to be not quite equal to it; he worked hard during the free hours to render himself able to perform his duty, but, being

over-conscientious, he was never satisfied with himself, and lost much strength. After having had a slight cough for a few weeks, he had a severe rigor on November 4th, and two days afterwards manifested distinct signs of pneumonia in the lower part of the right lung. The affection remained circumscribed; the constitutional symptoms were moderate. The fever reached its height on the evening of November 10th, when the temperature was 39°5 cent. (103°10 Fah.); pulse 115; there was no delirium; the urine contained scarcely a trace of albumen, and this had disappeared on November 12th, when the temperature at 6 p.m. was only 38° cent. (100°04 Fah.); the pulse 90. On November 14th, at 4 p.m., the temperature had descended to 37°5 cent. (99°5 Fah.), the pulse being 95 and weak. The pneumonic affection was in the best possible retrograde progress; the appetite good. Every symptom had appeared favorable, when at 4 a.m. on November 15th he became suddenly much excited, being under the impression that the house in which he was employed was on the point of being ruined through a mistake of his. When I saw him, at 9 a.m., he was quite maniacal, wanting to jump out of the window, having broken two panes of glass, having severely struck a near friend of his, and being with the greatest difficulty kept in his room by three persons. When for a few minutes somewhat calmer, he said that his employer and many other houses were ruined through him. Expression of extreme anxiety, and great collapse; face pale and cold; eyes sunken; hands and feet cold; perspiring profusely; pulse 105, very weak and irregular; temperature in axilla 37° cent. (98°6 Fah.). (One third of a grain of morphia every two hours; half an ounce of port wine every hour; to be kept quiet.)

At 2 p.m. he was still under the same delusion, but rather less violent; he had taken wine and food when offered, and had had two doses of morphia.

At 9 p.m., after six doses of morphia (two grains), the patient fell asleep, and slept till about 4 a.m. the following morning (November 16th), when he seemed to be still under

the same delusion; after another dose of morphia, however, he slept again for three hours, and in waking yielded to the representation that he could not have injured his friends by a mistake made during the last week, as he had been in bed for more than eight days. Pulse 84; temperature 36°5 cent. (97°7 Fah.); hands and feet warm; urine secreted in large quantity, forming a sediment of lithates. Expression of face composed, paleness less striking. Further progress satisfactory.

After *typhoid fever* I have met with two cases of this kind of delirium in the course of the last few months. The first of these (Case 6) I attended in the sanatorium of the German Hospital together with my friend Dr. Bacumber, the senior resident-physician, to whom I am indebted for the greater part of the notes.

Case VI.—L—, æt. 22, of rather excitable temperament and inclined to work beyond his strength, after having felt some amount of lassitude during several weeks, exhibited the distinct phenomena of typhoid fever in the first days of August, 1864; he had fever, headache, diarrhœa, rose-spots, enlargement of spleen, &c., all well marked, but not excessive; the symptoms of pyrexia culminated between the 6th and 8th of August, when the temperature varied between 28°5 and 40°2 cent. (101°3 and 104°36 Fah.), and the pulse between 90 and 98.

There had not been any delirium during this period. After August 9th the temperature never reached 38°5 cent. (101°3 Fah.). The patient received a moderate amount of stimulants, beef-tea and milk. On August 12th at 9 a.m., the temperature was only 37°5 cent. (99°5 Fah.); the pulse 78; no more diarrhœa; no fresh rose-spots. At 7 p.m. temperature 38°3 cent. (100°94 Fah.); pulse 88. During the following night he became suddenly delirious; he left his bed, went into the hall, and was on the point of quitting the house, when he was arrested and with some difficulty persuaded to return to bed; he fancied he had been called to perform some urgent business. After a little while he imagined he

was to appear before the coroner's jury in Müller's case (the murderer of Mr. Briggs); again after some time he believed that a young woman was bringing an action against him for breach of promise of marriage, which he never had given. On the whole, the subjects of the delirium were changing. At the same time the patient presented the appearance of collapse; the face pale; the extremities rather cold; perspiring freely. (Moderate doses of opium, wine, and beef-tea.)

At 9 a.m. on August 13th he was calmer, but not yet free from delusion; he gave, however, reasonable answers to pointed questions. Pulse 90, weak; temperature 37°1 cent. (98°78 Fah.). Towards the evening he slept occasionally, and during the following night, after half a grain of morphia, he had much sound sleep. On August 14th he was quite reasonable; the pulse at 9 a.m. was 74; the temperature 37°2 cent. (98°96 Fah.). No delirium returned after this time. The temperature remained still above 37° cent. (98°6 Fah.), therefore slightly above the standard, for six days, but the convalescence was not otherwise interrupted, and the recovery was perfect.

Case VII.—P. W—, æt. 28, a merchant, naturally anxious and excitable, otherwise healthy, and of temperate habits, had been in constant uneasiness for about two months on account of the frequent failures in the city and abroad. He had felt unusual heaviness in the limbs during the last days of October, 1864, and experienced a distinct rigor on November 2nd, and again on the evening of the 3rd. On November 9th, at 4 p.m., when I first saw him, there were unmistakeable symptoms of typhoid fever—moderate diarrhœa, and meteorismus; elevated rose-spots scattered over the trunk; enlarged spleen; red tongue, with tendency to dryness; bronchial catarrh; a frequent pulse, and a temperature of 39° cent. (102° Fahr.).

The treatment consisted in small doses of hydrochloric acid, beef-tea, and port wine (six ounces per diem).

The pyrexia increased during the following days, with slight evening exacerbations, the temperature varying from the 10th to 13th between 39° and 40°1 cent. (102°2 and 104°18 Fahr.); the pulse between 104 and 114; there was no delirium. From the 14th there was a marked decrease in all the morbid symptoms, and on November 17th (9 a.m.) the diarrhœa had ceased; there had not been any new spots for four days; the urine was free from albumen; pulse only 82; temperature 37°4 cent. (99°32 F.); the patient felt still weak; but took, in addition to his former allowance, a pint of milk, two eggs, and rather more wine. On waking on the following morning (November 18th) he became suddenly deranged, went almost undressed to the street door, said he had just been in the city, and must go again; he was ruined, and must stop payment; he heard people calling to him from the street, demanding him to arrange his affairs with them; he imagined, also, several times, that persons were in his room who were not there; he was very violent, and could scarcely be kept in the room by two strong men. He had a wild, anxious expression; his pale and cold face streamed with perspiration; pulse 110, very feeble, and rather irregular; hands and legs cold, temperature in axilla 36°4 cent. (97°52).

To be kept as quiet as possible; to have warm wine and beef-tea alternately every hour, and one third of a grain of morphia every three hours.

After ten hours of delirium, and after having taken four doses of morphia and about twelve ounces of port wine, the patient slept about six hours; on waking he still spoke of his failure, but after another dose of morphia he slept again for several hours, and on the morning of November 19th was quite reasonable, ascribing his delusion to a dream. The phenomena of collapse had likewise ceased; the hands and feet were warm; the pulse was 82 and stronger; the temperature 37°1 cent. (98°78 Fahr.). On the evening of this day, when the patient seemed again more excited, he took another dose of morphia, after which he had a good night, and went steadily on to perfect convalescence.

I have on record several other cases in which delirium suddenly manifested itself during the stadium decrementi, but, as they do not throw any additional light on the condition in question, it would be uncalled for to occupy the Society with their details.[1]

I have met also with mental derangement during and at the termination of *rheumatic fever*, different from the usual febrile delirium, but the condition of the patients was not of the same nature as in the cases under consideration. I will therefore at present abstain from entering on a discussion of the mental aberrations connected with rheumatic fever.

A careful examination of the cases above described will, I think, sufficiently show that the mental derangement in these patients is not to be regarded in the same light as the delirium usually observed during the increase and height of febrile diseases.

If we analyse the cases we find, with regard to the period at which the derangement occurred, that in all of them it manifested itself at the time when the pyrexia had almost ceased. Indeed, nearly all the medical men who have seen the cases with me said "There is no fever;" and to my own hand and eye the impression would probably have been the same, but the thermometer proved that the temperature of the blood

[1] Since the reading of this paper before the Society a very remarkable case occurred to me in a delicate and rather excitable man, aged twenty-five, who at the termination of a simple (not diphtheritic) inflammation of the tonsils and fauces, of rather more than average severity, suddenly was seized with mental derangement of a very gloomy character. Being under the impression that his prospects were hopelessly ruined, he intended to destroy, not only himself, but also his wife and infant child, in order to save them from future troubles. He was prevented from carrying out this intention, and recovered his reason in less than two days under the administration of morphia in large doses, and a moderate quantity of wine, but he remained rather desponding for more than a fortnight, after which he gradually regained his usual cheerfulness and mental energy. In this case, too, the phenomena of collapse accompanied the outbreak of the derangement.

was still higher than normal, with the single exception of Case 7, in which, soon after the outbreak of the delirium, the temperature in the axilla was found to be only 36°4 cent. (97°52 Fahr.), rising, however, again in the course of the day, and being on the following morning 37°1 (98°78 Fahr.).

The authors who have mentioned the subject have, generally, not given any thermometric figures, but restrict themselves to the expressions "during convalescence," or in the "commencement of convalescence." Griesinger,[1] however, mentions the temperature in two cases of pneumonia, in both of which the derangement commenced before the total cessation of pyrexia.

The pulse was in all the seven cases weak and rather frequent, in some irregular. The heart's action was repeatedly noted as feeble. It seems, however, from the observation of others, that increased frequency of the pulse is by no means necessary, at least if the derangement becomes protracted. Thus, Trousseau[2] mentions a case of delirium after typhoid fever in which the pulse was only 64, and in one of Griesinger's[3] cases of pneumonia it sank to 48 in the course of the derangement; one of the cases of delirium after maculated fever described by Graves[4] had a pulse of 60, or scarcely more.

Although the temperature of the blood was, as shown by the thermometric measurement in the axilla, in almost all the cases, still slightly increased, at least at the outbreak of the delirium, yet the hands and feet and the face were cold. The skin was in general perspiring profusely, and apparently on some parts of the body more than on others. The face was noticed to be pale, and the general appearance of the bodily condition, in spite of the mental excitement, was that of prostration or collapse.

The urine is repeatedly noted as having been passed in

[1] Griesinger, l. c., p. 191 ss.
[2] Trousseau, l. c., vol. i, p. 189.
[3] Griesinger, l. c., p. 191.
[4] Graves, l. c., p. 256.

large quantity, at least in the later period of the derangement or at the time of its cessation, and it was in all cases ascertained to be free from albumen and sugar.

The action of the bowels was regular, in all but two cases, in which it had been retarded for some days at the time of delirium.

The suddenness of the outbreak of the *mental derangement* is mentioned in all the cases, and the form approached, more or less, what may be termed maniacal delirium, with delusions of an anxious nature and hallucinations of the senses, especially of hearing, but also of sight. Nearly always there was a fixed delusion, but in Case 6 there was a repeated change in a comparatively short space of time, and in Cases 1 and 2 it changed likewise after an interval of sleep. The duration of the derangement was short, extending from less than eight to forty-eight hours. All these characters may be, however, by no means essential. Thus, although the maniacal delirium would seem to be the most frequent form, if judged from the preceding cases, yet there have been observed other forms, as dementia, especially by Sauvet,[1] after typhoid fever, and also monomania of grandeur ("monomanie des grandeurs") by the same author. The duration, too, appears to vary considerably, from a few minutes or hours to days and weeks and months.

The time of the outbreak was in general the early morning. As we may assume the seven cases to be equivalent to nine, because in two of them there were two distinct attacks, separated by perfectly free intervals, we find it particularly stated in six, viz. Cases 1, 3 (double), 4, 5, and 7, that the delirium began in the morning or soon after midnight (Case 4). In Case 2 the two outbreaks began in the evening; in Case 6 the hour is not specified, it being merely stated that the patient became suddenly delirious during the night. Almost always the commencement was stated to have occurred immediately after waking.

[1] Sauvet, l. c., pp. 223—230.

The subject of the delusion seems to depend, to some degree, on the subjects which have occupied the patient's mind a short time before the commencement of the illness. In many cases it would probably be difficult to trace this, but in Cases 2, 3, 5, 6, 7, it is very evident.

With regard to the condition of the brain and nervous system on which these derangements depend, or with which they are found connected, almost all the observers mentioned more or less agree in the view that it is "anæmia." The period of the disease at which the derangement occurs, the coldness of the hands and feet, the cold, pale, sunken face, the state of the heart and skin and of the whole body at the time of the occurrence, point in that direction. But "anæmia" is a general expression, applied to several or many more or less different conditions. Thus, there is certainly a difference between the "anæmia" caused by repeated losses of blood, or by long-continued insufficient ingestion of nourishment or excessive excretions, and the condition of the brain connected with these instances of mental aberration. In the former cases we have to deal with a more chronic alteration in the constitution of the blood, and in the consequent nutrition of the brain and other organs; in the latter, *i. e.* in the delirium or acute insanity under consideration, there is probably only a sudden and transitory change in the capillary circulation of the brain, and through this an equally transitory change in the nutrition and action of the brain-cells, a change which may be caused by a sudden sinking of the heart's power. In acute diseases the whole organism often experiences, at the time of the crisis, or more frequently some time after it, although occasionally also before it, a peculiar more or less sudden shock, which has been significantly called collapse.[1] Every

[1] The term "collapse" is used for the signification of different conditions. There may be a "collapse" from loss of blood, from excessive excretions, from the action of an animal poison, from sudden and excessive pain, from the shock caused by an injury, from a purely psychical influence, as grief or terror, and from other causes powerfully acting on the nervous system; but probably all these conditions differ more or less from each

one of us is now and then urgently summoned to see a patient whom he had left a few hours before in a condition which authorised him to say that the patient is in a fair way to recover. When we arrive we hear that "a sudden change has taken place;" that the patient is "sinking;" that the extremities have become cold, his face pale, his eyes sunken; that a shivering or a cold perspiration has come over him; that he can scarcely swallow any more; that the tone of his voice is quite altered. All these symptoms, and, in addition to them, those which we observe on the pulse and heart, may be more or less pronounced; they may soon pass off with or without treatment, or may last for some time and actually usher in death. These are the phenomena of "collapse," and to this condition it appears to me that the delirium or acute insanity in the cases above described is allied, and that it may therefore be called the *delirium of collapse*.

I am, however, inclined to think that not all the mental disturbances which appear during the decline of or convalescence from acute diseases are of the same nature. Thus, I have already alluded to a kind of mental derangement oc-

other, and also from the "collapse" in the sense here assumed, *i. e.* from the shock perceived by the system about the time or after the commencement of the crisis in acute disease. This shock is, perhaps, a perfectly normal occurrence in acute disease, but is in most cases so slight as altogether to escape attention, while in others it manifests itself only by a slight rigor, or a sudden sensation of weakness, or a transitory feeling of anxiety, or an unusual excitement, and only in few cases by the more alarming symptoms above described. Transitory, more or less perfect, failure of the power of the heart appears to be intimately connected with this remarkable condition, on which Wunderlich has communicated an excellent essay a few years ago ('Archiv. fur Heilkunde,' Jahrgang, 1861, p. 289).

Why in some rare cases delirium should occur as one of the phenomena of this species of collapse seems difficult to understand. Sleeplessness, pain, imperfect ingestion of food, and other influences impairing the nutrition of the nervous system, are, it is true, inseparable from the more serious acute diseases; but these alone, without some additional obscure cause, comprised in the term "predisposition," would scarcely account for the occurrence.

curring during and at the termination of rheumatic fever, which differs from the common febrile delirium, and also from the delirium of collapse.

I may refer to a very able essay on this subject by Griesinger,[1] whose views agree in most points with the results of my own experience, and also to a valuable article by Tüngel,[2] whose views, however, are somewhat different from those of Griesinger. Other instances, of a different kind of mental disturbance, may be seen in those cases where the delirium, which originated in the stage of increase or acme, is prolonged into that of convalescence. Again, others are those which begin more or less gradually during convalescence, are of longer duration, and are not attended with other symptoms of "collapse," although in these cases, too, an anæmic condition or an impaired nutrition of the brain may be inferred. Thus, I saw, in consultation with the late Mr. Huckey, of Wellclose Square, a female patient, about thirty years of age, who almost three weeks after the commencement of convalescence from a severe typhus (not typhoid) fever, and after she had been already several times out of doors, became depressed in spirits, and some days later represented herself, without any reason whatever, as ill-treated and persecuted by her friends, and attempted suicide by laudanum.[3] She was weak and anæmic, but quite free from fever. Under a tonic treatment, by rest, and a generous diet, she lost her delusion in a short time, and could afterwards scarcely conceive how it had been possible for her to misunderstand her friends and

[1] W. Griesinger, "Die protrahirte Form der rheumatischen Gehirnaffection." 'Archiv f. Heilkunde,' I. Jahrgang, p. 235, 1860.

[2] C. Tüngel, "Ueber die nervösen Zufaelle, bei'm atruten Gelents rheumatismus." 'Klinische Mittheilungen d. Hamburger Krankenhanses' von 1860, Hamburg, 1862, p. 99.

[3] A remarkable feature of this case was that the poor woman, who was quite unaccustomed to opium, had taken more than an ounce of laudanum, which was afterwards ascertained to be of average strength (viz. a drachm was equivalent to about three grains of opium) without fatal effect, but, on the contrary, apparently with advantage. Was this tolerance due to the peculiar condition of the brain?

to commit such an act. She mentioned, of her own accord, that at the time when she took the laudanum herself she had intended to kill her child also by the same means, in order to save it from future troubles, but had been prevented from carrying out her design by unexpected circumstances.

There occur also, sometimes, during the *increase* and *acme* of acute diseases, mental derangements which differ from common febrile delirium. Thus, I may mention the sad case of a strong male patient (under the treatment of my friend Dr. Lichtenburg and myself), who in the second week of typhoid fever became much depressed, and, during the night, committed suicide, by strangulation, while in his bed. Homicidal mania, too, may occur in acute diseases. But if it is at all periods difficult to draw a line of demarcation between what is usually termed "delirium" and what is recognised as "insanity," it is quite impossible to do so in the periods of increase and of acme of acute diseases. Collapse occurs also in these stages, although more rarely than during the decline, and this collapse may, perhaps, occasionally be attended with peculiar forms of delirium or acute insanity.

The preceding cases offer instances of the delirium of collapse in connection with measles, scarlet fever, pneumonia, erysipelas, and typhoid fever, but there is no reason to believe that it may not supervene on any acute disease, particularly in very excitable and anxious persons, with an exhausted nervous system, particularly if long deprived of sleep.

There seems to be no cause for attributing it to any particular morbid poison influencing the brain. It appears, also, that it may occur as well after diseases of moderate as after such of great intensity.

The prognosis may, in general, be considered as favorable, the derangement disappearing, mostly, in the course of a few hours or days, under the influence of proper treatment, without leaving any traces except those of a very vivid dream.[1] Graves calls it a "delirium of the most violent and dangerous

[1] Dr. Gairdner, in his allusion to the delirium in typhus arising after the crisis, regards the prognosis likewise as favorable.

description," but his patients seem to have recovered in a short time.[1] There are, however, many instances on record where the mental disorder commencing during the decline or convalescence from acute diseases became chronic, lasting several months or years; and the statistics of insanity sufficiently show that amongst the admissions into asylums many cases date their origin from acute diseases. In the excellent works on mental diseases by Griesinger[2] and by Bucknill and Tuke[3] we find fever, pneumonia, and other acute diseases, mentioned among the somatic causes of insanity. However, as already stated, I am not quite convinced that these chronic cases are not of the same nature as the delirium of collapse or of the decline of acute diseases. My experience on this point is very limited. It would be necessary to ascertain whether they originated in the stage of decline, or earlier or later; whether suddenly or gradually; whether the commencement was attended with other phenomena of collapse or not. They may, for instance, be caused by the general impairment of nutrition, and that of the brain especially, owing to the lowering effect of acute diseases, want of food, &c., and yet may not be due to "collapse."

The treatment adopted in the cases related to the Society consisted in the use of opiates, in rather large and frequently repeated doses, to allay the irritability of the brain, and in the simultaneous employment of means to counteract the collapse, viz. stimulants applied externally and administered internally, artificial warmth to the cold extremities, and food. The patients were, at the same time, kept as quiet as possible, and prevented from injuring themselves and those around them, and this, probably, in many instances would be sufficient, without any medicinal interference; but the opiates appeared certainly to accelerate the sleep and the return of

[1] Graves, l. c., p. 256.

[2] Griesinger, l. c., p. 179.

[3] 'A Manual of Psychological Medicine.' By John Charles Bucknill, M.D., and Daniel Hack Tuke, M.D. Second edition, London, 1862, p. 284.

reason, and they were borne remarkably well by the system, and might, perhaps, be administered in even much larger doses, if the smaller were not found efficient enough. Graves, too, gave opium, but in addition in some cases extract of belladonna in large doses and tartrate of antimony. Opium seems to possess also the approbation of Griesinger.

ON

INTERMITTENT HÆMATURIA;

WITH

REMARKS UPON ITS PATHOLOGY AND TREATMENT.

BY

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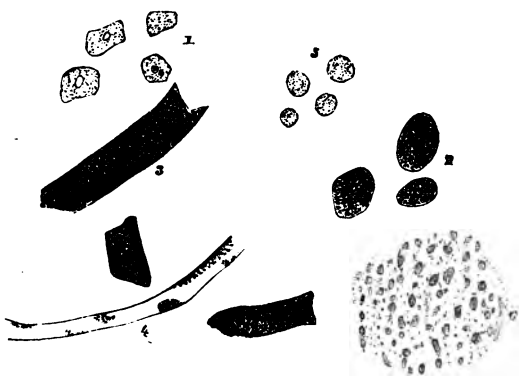
No more correct idea can be given of the special character of the disease about to be described than by quoting the reply of one of the patients when asked what was the matter with him. His answer was given in these words—"I can't tell you; but each time I get cold hands or cold feet I pass bloody urine, while my urine at other times is perfectly healthy."

In the other case the urinary symptom was not traced so much to the effects of cold as to malarial poisoning; but as it was the one which first fell under my observation, I shall consider it first.

Dr. ——, a member of our own profession, after several years' residence in one of the West Indian Islands, in consequence of repeated attacks of intermittent fever, was forced to give up practice and return to England, where for the

first two years he was still liable to occasional outbursts of his old enemy. On one occasion, while consulting me regarding his case, he mentioned what he considered to be a very peculiar symptom, namely, that he occasionally suddenly passed five or six ounces of urine of a dark red or chocolate colour, a symptom which would recur once in twenty-four hours during two or three days, and then as suddenly disappear. Never having before met with such a case, I requested him to send to me, on the next occasion, a specimen of the fluid. In the succeeding November (1861) I received from this gentleman three samples of urine—one passed at 8 a.m., which was clear, pale, of a specific gravity of 1025, of an acid reaction, depositing no lithates, and containing no albumen, being, in fact, normal in every respect. Another quantity, passed at 2 p.m. of the same day, of a dark chocolate-brown colour, opaque, turbid, having a specific gravity of 1032, of an acid reaction, depositing lithates, containing a large quantity of albumen, some sugar, and a large excess of urea (3·6 per cent.). The deposit from this specimen of urine, when examined with the microscope was found to contain nucleated epithelium (Fig. A, 1), some granular cells (Fig. A. 2), and a large quantity of free granules of a brownish-red hæmatin colour, scattered among which were a considerable number of renal tube-casts. The tube-casts presented one or two remarkable peculiarities, namely, that the majority of them were short and broad, and filled chokefull with brown pigment, as represented at Fig. A, 3. Besides these there were a small number of fine, long, pale tube-casts, with only a few granules of dark pigment distributed in them (Fig. A, 4); these looked not at all unlike the renal tubes emptied of their epithelium obtained by scraping a section of fresh kidney. No blood-corpuscles were to be found in this specimen of urine. The third sample of urine sent by the gentleman in question was passed in the evening of the same day, and presented a striking contrast to that just described. It was normal in colour, contained no albumen, deposited a small quantity of ordinary coloured lithates, among which were neither tube-casts nor granular cells. The specific

FIG. A.



1. Nucleated epithelium.
2. Large granular corpuscles.
3 and 4. Granular tube-casts.
5. Mucus-cells.
6. Free hæmatin and urates.

gravity of the liquid was 1021. Its reaction was acid, and its per-centage of urea exactly half (namely, 1·8 per cent.) of that of the preceding specimen. These three different conditions of the urine were certainly very peculiar; for had the morning's specimen alone been brought under the notice of the physician, he could never have dreamt of the existence of any urinary affection. On the other hand, if the single specimen of urine passed four hours later had been submitted to his inspection, he must have come at once to the conclusion that there existed grave organic changes in the renal organs. Whereas neither the one nor the other of these opinions could possibly be correct.

The gentleman alluded to at the time when he passed these urines was labouring under hepatic derangement, being, in fact, slightly jaundiced, as a result, most probably, of the malarial poison from the effects of which, as before said, he had not yet entirely recovered.

The varying conditions of the three urines clearly pointed to intense congestion of the chylopoietic viscera of a transient and periodic character. Suiting the practice to the theory, mercurials and afterwards quinine were taken by this gentle-

man, in order to remove the congestion of the chylopoietic viscera, and check the periodicity of the disease. The results were most favorable, for, although four years have passed away, he has never had a recurrence of these urinary symptoms. I shall now proceed to call attention to the second case.

On the 16th of December, 1864, M. N—, a dark, sallow-complexioned, careworn-looking man was kindly sent to me by my colleague Professor Fox, in consequence of his case presenting unusual characters.

The history of the patient is briefly as follows :—He is a blacksmith by trade, thirty-two years of age, and unmarried. Until two years ago he considered himself perfectly healthy, having always been able to do forge work without either difficulty or inconvenience, having, in fact, been a strong man.

Two years ago he for the first time observed that he occasionally passed urine as dark as brown old ale, while that voided at the preceding and succeeding micturitions possessed the normal colour and transparency.

Twelve months later, that is to say, a year ago, the urine for the first time assumed the colour of blood, a symptom which greatly alarmed him, as it recurred about three times a week during the whole of that winter, except during a fortnight in January, while working in the open air, when it became still more frequent, occurring about once every day.

Sometimes the attack of bloody urine lasted over two micturitions, amounting to a period of from four to five hours. In the spring of last year, as the warm weather advanced, the attacks gradually became less frequent, until from the month of May to September they entirely ceased. In September, however, they reappeared at intervals of about every ten days, the intervals gradually diminishing until a fortnight before he came under my care, when he passed bloody urine every other day, and for the last five days he has passed it every day at irregular hours varying between 10 a.m. and 6 p.m. The quantity usually emitted is about six ounces. The patient further stated that since the commencement of his illness, with the exception of the summer interval, he was

constantly under treatment by various medical men, without receiving the slightest benefit.

On his first visit to me the man brought with him two bottles containing the urine that he passed at 9 a.m. and at 2 p.m. on the previous day. The former sample was clear, transparent, straw-coloured, and normal-looking; the latter a dark purple blood-coloured fluid. On carefully cross-questioning the patient as to the origin of these liquids, he stated that the dark urine was usually passed about an hour after his feeling cold; that the urine did not invariably become clear at the next micturition: and that occasionally it did not reassume its perfectly natural colour until he had emptied his bladder three times. He stated, moreover, that he then felt cold, and that even during the time he was kept in the waiting-room he had passed four ounces perfectly similar to the bloody looking fluid which he had brought with him. He was accordingly requested to go behind the screen and make some more, which he immediately did, and produced about two ounces of a liquid of a dark purple-red colour. As he complained of feeling intensely cold, notwithstanding that he was sitting in front of a large fire, the temperature of the palms of his hands was taken, and found to be only 60° Fahr., while the temperature of my own hand was $95\frac{4}{10}$° Fahr. The temperature of the patient's axilla was also carefully taken with as little disturbance to his dress as was possible; and in spite of his being well clad with warm clothing, it was ascertained to be only $96\frac{1}{10}$° Fahr., a result which entirely confirmed his statement regarding his sensations of cold. He, moreover, added that he was a Londoner, and had never suffered from ague, the most that could be ascertained on this point being that on some occasions he had felt so cold as to shiver during the night, which shivering was not, however, followed by a true hot stage.

As before mentioned, the man was dark complexioned and had a sallow look; the sallowness, however, appeared to be due to some disturbance of the hepatic functions. He admitted that he was a very bilious subject, but denied having ever had any hepatic affection beyond what might be in-

cluded in the term functional derangement, but this had never at any time amounted to jaundice.

It will be observed that this and the preceding case present many features in common, the only apparent difference being that while the first could be distinctly traced to malarial poison, the second appears to be simply the result of the direct effects of cold acting upon a predisposed constitution. Such, at least, was the theory I formed of the disease; and, accordingly, the ordinary line of treatment recommended in hæmaturia was abandoned, and the plan adopted in the first case had recourse to, a course of treatment which proved most beneficial, for before twenty-four hours had elapsed the disease received a check, and by the end of forty-eight hours it may be said to have completely disappeared, for from that time until now, although fifteen weeks have elapsed, the patient has never had a single recurrence of his urinary symptoms.[1]

The amelioration of the condition of the patient in this case can scarcely, I think, be attributed to anything else than the effect of the treatment, as the diet and other conditions under which he was placed remained entirely unchanged. We cannot even suppose that the weather had anything to do with it, for the temperature of the atmosphere in the last two weeks of December, throughout January, February, and the beginning of March, was often lower than in any of the preceding months of this winter.[2] Moreover, until the very day on which the treatment was commenced the patient's condition had been gradually becoming worse and worse, while, as just said, within twenty-four hours after it was begun the disease had evidently received a check, and within forty-eight the urinary symptoms had entirely disappeared.

[1] The patient was a regular attendant at the hospital during the whole winter months, coming once a week, no matter how cold or wet the day was, up to the time the warm weather set in, when, by permission, he ceased his visits.

[2] The patient was at no time taken into the hospital, but made to come, as already said, once a week, no matter whether the day was wet or dry cold or fine.

At page 756, vol. i (third edition), of Dr. Watson's 'Lectures,' it is mentioned that when quinine, given alone, fails to cure an ague, a few grains of calomel, followed up with quinine, will often entirely check the disease, a fact which rather goes to support the view that even the second case might be due to a modified form of malarial poisoning.

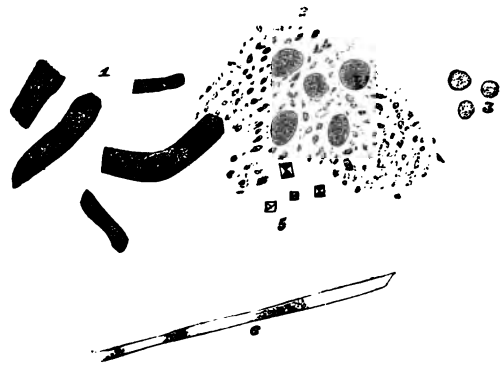
These notes might almost be allowed to end here; but as I consider that the pathology of such cases as have just been described is of great clinical value, I shall say a few words more regarding the condition of the urine, as by so doing it will not only be seen on what grounds I founded my diagnosis, but perhaps aid the labours of some future inquirer, who may have the good fortune to throw more light upon the nature of these cases than the data I have at present at command enable me to do.

The man was ordered to preserve all the urine he passed during the forty-eight hours after I first saw him, and to put what was passed at each micturition into separate bottles.

On examination it was found that the specimen passed at 8.30 a.m. was normal in colour, devoid of any sediment, six and a half ounces in quantity, acid in reaction, and of a specific gravity of 1010; it contained 1·75 per cent. of urea, traces of sugar, but no albumen.

That passed at 2 p.m. was dark red, almost black-looking, six ounces in quantity, acid in reaction, of a specific gravity of 1017, and on standing deposited a copious precipitate of dark-coloured urate of soda, leaving the supernatant liquid quite clear, and of a fine rich port-wine colour. This urine contained 2·5 per cent. of urea, was highly coagulable by heat and nitric acid, and gave evidences of traces of sugar. On examining the deposit from it under the microscope, although scarcely a single blood-corpuscle was to be found, it contained a great abundance of granular tube-casts (Fig. B, 1), large granular cells (2), free granular matter of a hæmatine colour (4), and among the urates a few octahedral crystals of oxalate of lime (5). It will be seen that this urine presents a striking resemblance, both in its chemical and micro-

FIG. B.



1. Granular tube-casts.
2. Large granular cells.
3. Mucus-corpuscles.
4. Free hæmatin.
5. Oxalate-of-lime crystals.
6. Fine hyaline tube-cast.

scopical character, to that of Dr. —, being of high specific gravity, coagulable by heat and nitric acid; containing a large excess of urea, abundance of granular tube-casts, and scarcely any blood-corpuscles.[1]

The urine passed at 6.30 p.m. was five and a half ounces in quantity, slightly smoky in colour, with a moderate deposit of pale urate of soda, a specific gravity of 1016, and acid reaction; it contained 2 per cent. of urea, and was slightly coagulable by heat and nitric acid. The deposit, when examined by the microscope, was found to contain only a few granular tube-casts, one very long one (Fig. B, 6), with some granules in it; a few mucus-cells, no crystals of oxalate of lime, no blood-corpuscles, and only small collections of pigment scattered among the amorphous urate of soda.

The urine passed at 9 p.m. was about six ounces in quan-

[1] For all practical purposes one may say that there were no blood-corpuscles in the urine, for out of sixteen of the gentlemen attending my practical class only two found any, and even then there were only one or two in the field of the microscope; while, had the case been one of ordinary hæmaturia, the absence of blood-corpuscles would have been the exception, not the rule.

tity, perfectly normal in colour, without any sediment; had a specific gravity of 1016; acid reaction; contained 1·84 per cent. of urea, and not a trace of albumen.

The urines passed on the following day were all normal in colour except one, namely, that passed at 10 a.m., which was of a slight brownish-red tint; it deposited a copious sediment, coagulated freely on the application of heat and nitric acid, and contained 2·05 per cent. of urea. In the sediment of this urine were found a number of granular tube-casts, but nothing like the quantity in that of the well-marked specimen. This specimen presented, in fact, the last appearance of the disease, for from then until now the urine has never again become either albuminous or of a dark colour, and only twice, on the 12th and 20th of January, has it deposited any precipitate. On the first occasion the patient suspected a recurrence of his old disease, a suspicion which, fortunately for him, was not verified, for, on testing the urine, it was found to become perfectly transparent on the application of heat, and to be entirely devoid of albumen or tube-casts, the turbidity being simply due to the presence of urate of soda. The second specimen was exactly similar.

There are one or two points regarding the condition of these various urines requiring special notice.

1. The presence of the granular tube-casts clearly points to congestion of the renal organs, but their appearing and disappearing in the course of a few hours at the same time, proves that it was not an ordinary case of renal congestion.

2. The almost total absence of blood-corpuscles, notwithstanding the hæmorrhagic appearance of the urine, stamps the case as being entirely different from ordinary hæmaturia, and shows its resemblance, in this particular, to that variety of non-intermittent hæmaturia in which the contents of the blood-corpuscles alone pass into the urine.

3. It cannot be regarded as a sample of simple intermitting albuminuria; for although the protein substance coagulable by heat and nitric acid has been hitherto spoken of as albumen, it differed very materially from the albumen of

blood-serum in its ready solubility in an excess of acid. In making the analysis it was found necessary to be exceedingly careful with regard to the amount of nitric or acetic acid employed, for after the coagulable point was arrived at the addition of a single drop more of either of these acids instantly redissolved the coagulum, and set the colouring matter free, a circumstance which, taken in conjunction with the uniform diffusion of the pigment and the coagulable matter in the liquid, leads to the conclusion that it was not simply the albumen of the blood-serum, but the hæmato-globulin itself, which was excreted by the kidneys.

4. The case was not one of urohæmaturia, such as I have elsewhere described,[1] for two reasons—first, on account of the urine being coagulable by heat and nitric acid; and, secondly, from the fact of the addition of acids diminishing instead of increasing the dark tint of the urine.

5. The bilious appearance of the patients in both cases would lead to the belief that the attacks were in some way or another connected with the disturbance of the hepatic function, which, at least in one case, was distinctly traceable to malarial poisoning.

6. The copious deposition of urates, as well as the excessive elimination of urea, which takes place during the attack, points to considerable general constitutional disturbance.

Lastly, the transitory condition of the urinary symptoms show that, whatever might be the nature of the disease, the exciting cause could not be in constant operation, unless we admit that it required distinct periods of incubation, as in ague, which we know is liable to assume an almost endless variety of forms. In conclusion, I have only to remark that it is of great clinical importance to be able to make a correct differential diagnosis between cases like the present and those of ordinary hæmaturia, for without it it will be utterly impossible to treat them with much chance of success.

[1] "Course of Lectures on the Urine, and Diseases of the Urinary Organs;" 'Med. Times and Gaz.,' September, 1864.

The last quoted case is, indeed, a striking illustration of the truth of this remark, for, notwithstanding the patient having been at different London hospitals, under the care of men of high professional standing, he failed to obtain relief, in consequence of the orthodox line of treatment laid down for ordinary hæmaturia having been adopted.

APPENDIX.

Although many urologists have referred to cases of intermittent hæmaturia, Rayer, as far as I can find, is the only one who has made any special mention of this particular subject; none of the cases which he relates, however, seem to have fallen under his own observation. The cases he alludes to are the following.

Case 1.—On the 24th November, 1832, a man suffering from diseased heart and ague came into University College Hospital, under the care of Dr. Elliotson. "The singular circumstance, however, in this rare disease was, that when his paroxysms came on he discharged bloody urine . . . . . at first pure blood, and afterwards less and less, and this he said was invariably the case—hæmaturia every time the cold fit came on. This circumstance, however, made no difference in the treatment, and I gave him . . . . . ten grains of sulphate of quinine three times a day till he became perfectly well, so far as his aguish symptoms were concerned. He lost the rigors, he lost the cold fit, and he lost the bloody urine.

"The bloody urine was intermittent, like the rigors; that is an interesting circumstance; I never met with an instance of a similar description. There can be no doubt of its truth, because the man showed his urine, and the blood was abundant in it. He was presently quite well so far as this was

concerned, and the symptoms arising from hypertrophy of the heart were much diminished. Having had aguish fever, however, in the severe form, which he suffers whenever the east wind blows or he is exposed to cold and wet, or commits any error in diet or is guilty of any debauchery, he will be liable to a return of the disease."[1]

Case 2.—"M. Gergerés was called to a young man, a captain in the navy, who generally enjoyed good health, but who for two hours had been seized with severe shivering fits, which were followed by great heat; during this period the patient felt a desire to make water, but, instead of urine, he passed a large quantity of blood. A few hours after, an improvement took place, and the patient thought himself cured. On the following day, at the same hour, the same feverish symptoms occurred, accompanied by the elimination of blood. M. Gergerés prescribed an emollient treatment, both internally and externally. The symptoms disappeared again at the same hour as on the first attack. On the third day the same phenomena appeared, with even more violence. From this time, towards the end of the attack, a dose of twenty-five grains of quinine was administered, and this method prevented any return of the symptoms."[2]

Case 3.—(The original report of this case I have not been able to refer to.—G. H.) "Stewart[3] had a patient labouring under a periodic hæmaturia, which was unsuccessfully treated antiphlogistically during eight months, and which disappeared after a further three months' course of quinine and tonics."

In none of these cases, unfortunately, is any mention made of the chemical constitution or microscopic appearances presented by the fluid passed; and although it is just possible, from the circumstance of Dr. Elliotson employing the term "pure blood," that the case he describes might be one of the

[1] 'Lancet,' 1832, p. 500.
[2] 'Gazette Médicale de Paris,' March, 1838, p. 151.
[3] 'Rayer,' vol. iii, p. 370.

ordinary forms of intermitting hæmaturia, I think it highly probable that it is the exact counterpart of those I have described, and that the term "pure blood" is due to a mistake arising from the imperfect means of observation existing at that time. We must not forget that the microscope was comparatively little heard of three and thirty years ago.

However, this is a point scarcely worth discussing, for it is to the cases of the future, not to those of the past, that we must look to for the true explanation of the pathology of this disease. And I have little doubt that we shall not require to wait long before the subject is fully elucidated, for, now that some of the more striking features of the urine have been pointed out, such as the presence of granular tube-casts, the absence of blood-corpuscles, the increase in the amount of the urea, &c., plenty of similar cases will soon be diagnosed and reported.

NOTES OF FOUR CASES

OF

INTERMITTENT HÆMATURIA.

BY

WILLIAM H. DICKINSON, M.D. CANTAB.,

FELLOW OF THE ROYAL COLLEGE OF PHYSICIANS; CURATOR AT ST. GEORGE'S HOSPITAL; ASSISTANT-PHYSICIAN TO THE HOSPITAL FOR SICK CHILDREN.

Received March 7th.—Read May 9th, 1865.

THE man who is the subject of the following report has often been under observation at St. George's Hospital, where the characters of his complaint have become well known. The notice of Dr. Harley's cases was the means of suggesting that the particulars of another might be interesting to the Society, and at Dr. Fuller's request I have endeavoured to supply them. It will not be necessary to refer to any preceding accounts of this affection. Dr. Harley has, no doubt, given sufficient details on this head to show that, though of rare occurrence, the disorder has not escaped notice.

George M—, æt. 40, is now in St. George's Hospital under the care of Dr. Fuller; he is a builder's labourer, employed chiefly in the open air, and necessarily exposed to weather. He had an attack of tertian ague when working at Whatton-in-the-Vale, in Lincolnshire, more than twenty years ago.

He recovered under medical treatment, without, as far as he knows, having experienced any change in the character of the urine. His health after that time remained good until the autumn of 1859, when he was employed in building the Broadmoor Asylum in Berkshire. At the end of October or the beginning of November, the weather at that time being very cold and damp, he was first attacked with the disorder to which he has since been subject. It began one morning with shivering, ineffectual attempts to vomit, and dull pains in the loins. When he passed water he found that it was black and apparently bloody. He learnt from a medical practitioner to whom he applied that ague was prevalent in the district, but he had no symptoms of that disease except the shivering. There was no hot stage. He left his work and got employment in London, and from that time to the present he has been often under observation at St. George's Hospital. He was first under Dr. Ogle, as an out-patient. He has since been in the house at different times, under Dr. Bence Jones, Dr. Pitman, Dr. Fuller, and Dr. Barclay, six times in all. Avoiding repetition, the general characters of his complaint may be briefly stated.

He has no constant ailment, but his health is interrupted by short attacks of hæmaturia. From the beginning of the disorder these have always been of the same character, though at first they were less severe than they afterwards became. They are brought on by no other cause than exposure to cold, and usually happen in this manner:—He gets up and goes to his work apparently well. If he chance to be exposed to cold, he has an attack of shivering and retching; he yawns, and is disposed to stretch himself; he has heavy pain in the loins, passing down the thigh, and both testicles are closely retracted. Within an hour or so he has an inclination to pass water, which is voided in considerable quantity, and is black, like porter, and turbid. The pain in the loins, which has been on the increase, now slowly subsides, and the constitutional disturbance ceases. When the attack comes on he leaves his work and goes home to bed, taking care to be very warm. Under these circumstances the urine

retains it character for two or three times of passing, and then resumes its natural appearance. The attack altogether lasts from three to twelve hours. It is often followed in the evening by pain, which is described as of a griping or colicky sort, about the umbilicus, where he states that he can sometimes feel a hardness, which passes off with the pain. Next day the only remnants of the complaint are the weakness and pallor which result from the loss of blood. I will now give a more particular account of the urine.

The urine passed during the attack has been frequently examined, and has always been found to present the same character. It is nearly black and turbid. After standing, a black powdery deposit is thrown down, leaving the upper stratum clear and of a dark reddish-brown colour. It is acid, and generally above 1020 specific gravity. Dr. Gee, who has given much attention to urinary analysis, was kind enough to examine some urine passed during a recent attack, February 20th. His account of it is as follows:

"Specific gravity 1023; much albumen; the dried coagulum has a lake colour, which it gives up to alcohol and sulphuric acid.

"Colour almost opaque, in a stratum an inch thick, at least as dark as the ninth tint of Vogel, "brownish-black."

"Urea 2·35 per cent.; chloride of sodium ·45 per cent."

The urea being rather above the average, but quite within the limits of normal variation.

Dr. Harley, who, I believe, first pointed out the increase of urea in these cases, has kindly supplied me with another estimation from a sample of urine passed during a later attack. The urea was in the proportion of 4·25 per cent., which is a decided excess.

Between the attacks the urine is natural. It contains no albumen, and nothing abnormal is to be seen under the microscope. It is always acid, of low specific gravity, varying from 1009 to 1017, lowest immediately after the seizure. The colour is rather deeper than usual. Dr. Gee, who ex-

amined twenty-four hours' urine—a quantity not to be obtained during the attack—gives the following details.

		Average of health.
"Quantity in cubic centimètres .	1728	1500
Specific gravity . . .	1014	—
Urea (grammes) . .	27·65	33·18
Chloride of sodium . .	8·64	11·50
Phosphoric acid . .	2·59	2·0 to 3·0
Pigment (Vogel's standard) .	13·0	5·0

"There was a slight reaction of indican. There was no trace of albumen or sugar. The most noteworthy fact about the urine was the excess of pigment, notwithstanding the diluteness of the fluid. The tint was, more brown than yellow, and remained so in spite of further dilution. It nearly corresponded with Vogel's fourth tint, 'reddish-yellow.'"

On one occasion, when the man had an unusually brief attack, I had an opportunity of observing the sudden change in the nature of the urine. The hæmaturia lasted but for one discharge. The urine then passed was of a dark brown colour, not black, as it has generally been. It was small in quantity, acid, specific gravity 1025. The microscope showed, as it always has done under the same circumstances, abundance of fine granular matter, with dark granular casts. When boiled the albuminous clot occupied a third of the bulk of the fluid, which had a rich transparent brown colour. The urine passed at the very next time was copious and watery; specific gravity 1009. It did not contain a trace of albumen, nor did the microscope show any corpuscles or deposit of any kind. It was acid, like the other.

The attacks which have been described have never continued through the night, though they have come on for several successive days. Sometimes, in continuous cold weather, he has had it three times a week for five or six weeks. He has never been seized when in-doors, even during the summer, though once it was brought on in comparatively warm weather by his washing windows in cold water. Movement has no tendency to cause it; in fact, he is better when

taking active exercise, because it keeps him warm. The earlier attacks were attended with less loss of blood than has accompanied the later seizures. About four years ago they attained their greatest severity, and were brought on with the greatest readiness. He has since been getting better in both respects. He has never passed any sand or fragments of stone, and, indeed, the history of the case is enough to show that the symptoms do not depend upon a calculus.

Whenever he has been admitted into the hospital he has had an anæmic and cachectic look. There is no permanent pain or tenderness about the loins, nor is there any other complaint of a persistent kind. The liver and spleen appear quite natural in size. The sounds of the heart are normal.

I need not dwell upon the treatment which has been adopted. Many experiments have been tried on this head. He has been cupped on the loins—has had vapour baths—he has taken quinine with perseverance, as well as gallic acid and iron in various forms; but as yet nothing has been found to affect the disorder. The hæmorrhage has always ceased on the removal of the cold which produced it, and has recurred with undiminished readiness on the next exposure.

Since the paper was sent in another experiment has been made as to treatment, which I have been allowed to insert. On the 22nd of February, when he was last admitted, Dr. Fuller determined to test the mercurial treatment, of which he had heard favorable reports. At that date, as always during the intermissions, the urine was natural, and the patient, though anæmic and sallow, was without any definite complaint. He was now ordered to take three grains of blue pill, with a quarter of a grain of opium, three times a day.

On the 2nd of March the pill was prescribed twice instead of three times a day, and a quinine draught was ordered also twice a day.

Two days later his gums were affected by the mercury, and the pill was continued only once daily, which sufficed to keep up the soreness of the month.

On March 8th, while taking the mercurial as described, and six grains of quinine daily, he had a shiver, with pain in the back, retraction of the testicles, and retching, but without any change in the appearance of the urine. This was the first time that these symptoms had come on without hæmaturia.

On the next day but one he was attacked in the same way, but now the hæmaturia followed according to his usual experience. He had been confined to the ward. This seizure was the first he had ever experienced in-doors.

On the 14th the mercurial medicine was discontinued.

On the 18th he had a rigor, followed by headache, and the next day by a stitch in the left side and symptoms of pneumonia. He had cough, with expectoration of bloody mucus, without any marked dyspnœa. The left side of the chest was dull on percussion, particularly towards the base. The vocal fremitus could be felt to the bottom of the lung, so that it was clear that the dulness was not due to the presence of fluid, though at one time pleural frictions had been audible. The voice-sounds were bronchial, and some coarse pneumonic crepitation was heard. The patient attributed this attack to his having, as he supposed, caught cold in the hospital chapel. An ammoniated saline draught was given, and afterwards some antimonial wine. It now was found that there was a great excess of urea in the urine, the addition of nitric acid converting the whole into a mass of crystals.

The urea on the 23rd was found to amount to 4·8 per cent., and on the 28th to 4·6 per cent. This peculiarity lasted for nearly a fortnight, the specific gravity ranging from 1024 to 1028. At the same time there was a minute trace of albumen. It is known that this state of urine is the frequent result of pneumonia.

The lung symptoms passed off, but the man grew hollow-visaged and cadaverous; the pulse very feeble and rapid (120—130), the tongue brownish and dry. He wandered in his mind, and then passed into a typhoid condition, lying on his back prostrate and speechless, though he retained a look of intelligence, and would make tremulous attempts to put out his tongue

when requested to do so. The evacuations were passed into the bed. He now, under the action of wine and nourishing diet, mended somewhat, and on the 30th had a sort of misguided activity about him, often trying to get out of bed; the pulse was now very rapid (144), and as feeble as ever; he was still speechless, and somewhat delirious, but could make known his wish to pass his evacuations. From this time under stimulating treatment he slowly improved, and at the same time the amount of urea diminished.

On April 6th he had recovered his usual manner and state of mind, and his pulse and tongue were natural, but he was much emaciated. The urine was now natural in all respects; specific gravity 1016; by the end of the month, under the use of quinine and iron, he was enabled to leave the hospital, weak and thin, but otherwise in his usual state of health.

Owing to the kindness of Dr. George Johnson, I am enabled to give the outline of two similar cases which have come under his observation. Dr. Johnson's notes are as follows:

"George K—, æt. 24, a photographer, living at Chatham, admitted into King's College Hospital under my care, January 7th, 1865. A man of intemperate habits. For three months before his admission he had been subject to attacks of hæmaturia, unattended with pain, and usually excited by a chill. The patient is still in the hospital, and during his stay there the hæmaturia has recurred several times. So long as he remains in bed the urine is free from blood, and does not coagulate by heat or acid. If he sits up and becomes chilled he has a feeling of coldness and general malaise, followed by heat of skin and sometimes by sweating, and the urine now passed has the colour of porter. On microscopic examination numerous tube-casts, apparently composed of disintegrated blood, are visible, with much scattered brown granular matter; few, if any, blood-corpuscles."

"Mr. D—, æt. 43, consulted me on the 19th of January, by the advice of Dr. Woollett, of Monmouth. This gentleman has had several attacks of hæmaturia, induced always by exposure to cold. The first attack, as I learn from Dr. Woollett, was in December, 1862; then on the 7th and 10th of January, 1863.

Two attacks in January, 1864. Again in December, 1864. Three or four attacks since then. When he called on me, in January last, he had the appearance of robust health. His urine was then perfectly normal, as it has always been when free from blood. The following day he went home to Monmouth by rail; on the journey he got a chill, and when he reached home he passed some urine, which he sent back to me. It was deeply tinged with blood, and contained numerous tube-casts, apparently composed of disintegrated blood, with much scattered granular matter of a brownish-red colour. There were few, if any, entire blood-corpuscles. This gentleman always knows when an attack is coming on. He feels cold, languid, and miserable, with a continual tendency to yawn, but he has no pain. The urine now has the blood tinge; that next passed is usually less coloured, and in the course of a few hours the urine has resumed the natural appearance."

Dr. Frederick Cock, of Westbourne Park Terrace, has obliged me with the particulars of a fourth case, which has been more or less under his observation for eleven years.

The patient is a country gentleman, about thirty-two years of age. The complaint began eleven years ago, with an attack of hæmaturia, which lasted several weeks, and then left him free for an interval of more than two years. Since this attack the symptoms have occasionally recurred; last spring there was an attack of unusual severity, since which the patient has been almost free from the complaint. The hæmaturia is always preceded by what is described as a "severe cold"—shivering, headache, nausea, pains in the limbs, and a twinge in the loins on stooping. After these symptoms have lasted for two or three days the urine assumes a dark colour, like London porter; it froths readily, and is highly albuminous, the coagulum occupying half the bulk of the urine in the test-tube. Under the microscope Dr. Cock has found a few uric-acid crystals and a very small quantity of mucus. No blood-corpuscles have ever been detected, but there is a yellowish molecular deposit, apparently the result of their disintegration. The dark colour of the urine generally dimin-

ishes, until in a fortnight or three weeks it has its natural colour and transparency. The albumen diminishes simultaneously, until only a very minute trace remains, which in its turn passes away. The microscopical appearances of the urine in the intervals are natural, excepting that a few uric-acid crystals are generally present.

This gentleman always traces his attacks to cold, and is particularly liable during the prevalence of east winds. He is seized more often in the spring and autumn than in the winter; never in the summer.

He remains very anæmic, and has a slight systolic murmur at the base of the heart, believed to be functional. He has never had ague.

He has been left almost without medical treatment. He is able to take active exercise and to hunt without inconvenience.

As to the pathology of the disorder, the cases are as yet too few to allow much to be said. From the abundance and character of the casts it is clear that the blood comes from the kidneys. From the small size of the cylinders and the rare occurrence of renal epithelium in them, it must be inferred that the blood flows from the Malpighian bodies, while the tubes retain their natural lining. The rapid restoration of the natural characters of the urine, and its total freedom from albumen in the intervals, would seem to suggest that there is no rupture of the vessels. If this be allowed, we must conclude that the disintegration of the blood-corpuscles has taken place within the vessels, and not subsequent to extravasation. We may believe that the impalpable amorphous material may transude without any opening such as would be needed to give exit to the entire corpuscles. Under these circumstances the disease must be regarded rather as connected with the blood than of primarily renal origin. We know that the same state of urine has been observed during typhus, and that it has followed, both in man and animals, the inhalation of arseniuretted hydrogen (Neubauer and Vogel 'On the Urine,' p. 310).

There are some points which the disorder has in common with

ague. The rigors, the yawning and stretching, the epigastric pain, the increase of urea during the attacks, are points of resemblance to the cold stage of an ague fit; but in only one of the four cases had there been ague in its usual form, and then it was obviously distinct from the hæmaturia. The attacks in all the cases were without any periodical tendency, and where the experiment was tried they were unaffected by quinine.

In the present state of our knowledge we must be content to regard the disorder as something *sui generis*, and assign the blood itself as the seat of the primary change.

There is little reason to believe that any treatment can do more than mitigate the effects. It was perfectly evident in the case of M— that the recurrence of the hæmorrhage was not retarded by any of the medicines he took, though sometimes he went for a long time without any attack. The symptoms which followed the use of mercury were not such as to encourage a repetition of the experiment. It will be remembered that while under its action he had, for the first time, an attack without leaving the house. Considering the anæmic state to which these patients are reduced, it would seem unwise to give them a drug of which one effect is to produce that condition. The variable length of the intervals between the attacks—in one instance more than two years—must make us cautious in attributing their absence to medical treatment.

SUPPLEMENT TO A PAPER

ENTITLED

FURTHER OBSERVATIONS

ON THE

STRUCTURE AND TREATMENT OF UTERINE POLYPI,

PUBLISHED IN THE XLIV VOLUME OF THE 'MEDICO-CHIRURGICAL TRANSACTIONS.'

BY

ROBERT LEE, M.D., F.R.S.

Received Feb. 10th.—Read May 23rd, 1865.

The paper to which reference has been made concludes with the following statement:

"By an examination of this condensed history of 105 cases of uterine polypi it will be seen that of the last 50 cases which have come under my own care, and in which the operation for the removal of the polypi has been performed by me, not one patient has died. The various methods of treatment, having been minutely described in the histories of the cases, do not require to be again detailed.

"Being fully aware that the greater number of large uterine polypi are fibrous tumours covered with the lining membrane and a portion of the muscular coat, and that

these polypi have large arteries and veins distributed throughout their substance, in operating upon them I have not ventured to drag them out of the vagina with forceps of any kind and divide their roots with the knife. The ligature has usually been applied in such cases with the bent rod, and the sloughing polypus has been removed when the ligature was long in dividing the root. By this means all the evil consequences which could be produced by the vagina being long filled with a putrid mass have been completed avoided, and to this course I do think may be attributed, in a great degree, the uniform success of operations performed by me of late. Even where the polypi have been of comparatively small size, and the ligature has been applied with the double canula, I have not considered it safe to leave the ligature many days around the root of the polypus when the circulation through the substance had been destroyed and the mass of the tumour was in a sloughing condition. By twisting the canula firmly round, the ligature has frequently passed through the root, and the polypus has been removed several days before this would have happened if the ligature had been merely tightened. Even in cases where the root of the polypus has not yielded to this treatment the ligature has been removed and the dead polypus has come away harmlessly after some days, tepid water having been freely injected into the vagina. In all cases it has appeared to be of the utmost consequence to watch the condition of the uterine organs and of the general system, as it has been clearly proved that inflammation is the most common cause of death after the application of a ligature around the peduncle of a polypus."

Since the publication of this paper in the 'Medico-Chirurgical Transactions' fifteen cases of uterine polypi have come under my observation. Three of these, which were of the most formidable character, from the great magnitude of the tumours and the impossibility of reaching their roots or the os uteri with the finger, were tied by a ligature passed up around them as high as practicable with the double canula. When the vitality of the polypi was destroyed and they were in a

sloughing state they were forcibly drawn out of the vagina with different kinds of forceps and hooks, and their roots divided with a knife or scissors if they had not previously been divided by the ligature. Of these fifteen cases contained in this table not one died. If these fifteen be added to the fifty cases previously reported it will appear that of sixty-five cases of polypus of the uterus which have come under my care, and been operated upon by me, not one has died.

Thirteen of the cases now reported occurred in Burton Ward, St. George's Hospital. For the tabular view of these cases I am indebted to Mr. Smith, late obstetric-assistant, who has filled the office during two years in the most efficient manner.

A Tabular View of Fifteen additional Cases of Uterine Polypi.

	Date.	Name.	Age.	Social condition.	Symptoms and treatment.	Result.
106	October 24th, 1861	Sarah S—, Burton Ward, St. George's Hospital	55	Married; 11 children; 8 miscarriages	Profuse hæmorrhages for twelve months; anæmia; pain in back and hypogastrium; a pyriform tumour, the size of a pea, felt projecting through the os uteri. The ligature applied by means of the double canula on the 29th. No unpleasant symptoms; the tumour came away with the canula on the 31st. Discharged November 5th.	Recovered.
107	January 8th, 1862	Ellen A—, Burton Ward, St. George's Hospital	40	Married; 5 children	Suppression of menstruation, with pink-coloured discharge, for ten months; abdomen swollen and tympanitic; mammæ rather large and containing milk; bearing-down pains. On examination the os was found open and a smooth body felt at upper part of cervix. She was kept in bed; the discharge ceased, the os uteri closed, and the tumour was never felt again. When last seen (May, 1864) she was quite well, and catamenia regular.	No operation performed.

108	January 22nd, 1862	Anne H—, St. George's Hospital	39	Married; 8 children; 1 miscarriage	Miscarried four months ago; profuse hæmorrhage for a week, since then bearing-down pains, vomiting, and profuse watery discharge; great pallor and debility; a pyriform tumour, about the size of a large pear, was found projecting through the os uteri. With rest and generous diet her health improved, and on February 22nd a ligature was passed round the root by means of the double canula; urine drawn off for two days. No unpleasant symptoms; on the 24th the polypus and canula came away. Discharged March 12th, still very weak.	Recovered.
109	March 5th, 1862	Anne H—, St. George's Hospital	44	Widow; 1 child	Profuse and frequent menstruation for twelve months; debility; no pain; a small polypus, about the size of a nut, with a small pedicle, felt projecting through the os. An attempt was made on the 12th to pass a ligature round the root, but it was too fine; the tumour was then seized with a pair of forceps and removed by torsion; some pain felt; no hæmorrhage; aching pain in limbs and body for three days; no fever. Discharged April 1st. She is now (October, 1864) perfectly well.	Recovered.
110	March 15th, 1862	Eliza W— St. George's Hospital	43	Married; 2 children	Profuse menstruation for four years; bearing-down pains; great debility; a polypus the size of a large orange felt projecting through os uteri. April 1st the ligature was applied with some difficulty; the ends of the canula were within the os. On the 4th the canula was removed, the ligature left; slight feverish symptoms. The polypus came away on the 7th; it was not fibrous; some parts of it were very hard, cutting like cartilage. Discharged April 21st.	Recovered.

CASES OF UTERINE POLYPI—*continued.*

	Date.	Name.	Age.	Social condition.	Symptoms and treatment.	Result.
111	June 3rd, 1863	Maria K—, Burton Ward, St. George's Hospital	23	Married; 4 children	Confined prematurely a month ago; child putrid; since then profuse hæmorrhage and severe bearing-down pains. Examined June 9th, a large polypus found, soft and irregular; pedicle pretty thick, extending within the os; the pains and discharge ceased. On the 20th the polypus could not be found; the os was somewhat open July 1st; menstruated regularly twice, then became pregnant, and miscarried January, 1864. Examined March 29th; much hæmorrhage; no polypus felt; os nearly closed; anterior part of uterus hard and enlarged.	No operation performed.
112	Sept. 23rd, 1863	Louisa K—, Do.	49	Married; 7 children	Fifteen months ago menstruated profusely, and a small tumour projected from the vagina; pain and irritation in vagina; yellow discharge; catamenia regular; os uteri low down. A round fleshy tumour, about three inches in length, and not so thick as the little finger, projected from the anterior lip through the ostium vaginæ, covered with mucous membrane; very vascular and insensible. On the 29th a ligature was applied round it about half an inch from os; aching pain in pelvis and legs for many hours; the tumour and canula came away on 30th. October 13th, anterior lip of os thick; two small excrescences felt on it. Discharged on 15th; still some slight discharge, with smarting pain in vagina.	Recovered.

113	Sept. 30th, 1863	Maria P—, Do.	53	Widow; 5 children	A small polypus removed by torsion five years ago (No. 99); the hæmorrhages soon returned, with much yellow discharge; cutting pains in hypogastrium. A polypus two inches in length, and hardly thicker than whip-cord, attached to inside of cervix on left side. On October 10th a good portion removed by forceps; it came away piecemeal; a good deal of hæmorrhage. On the 24th a little more was removed by the forceps. On the 28th rigors; tenderness in abdomen; fever; recovered in about a fortnight. A small portion of polypus still left; the hæmorrhages and discharges did not recur. She was discharged December 2nd.	Relieved.
114	Sept. 30th, 1863	Florence L—, Do.	40	Single	Profuse menstruation for five months; a tumour protruding from vagina for one month; yellow discharge. A small polypus found protruding from ostium vaginæ, oval, not hard; long diameter one and a half inch; surface very vascular; pedicle over two inches in length, quarter of an inch in diameter. A ligature applied on October 3rd; no bad symptoms; polypus and canula came away on 5th. On the 15th discharged cured.	Recovered.
115	October 7th, 1863	Susan C—, Do.	30	Married; 2 children; 5 miscarriages	Profuse and frequent menstruation for eight months; watery discharge; great debility; pain in back; anæmia. In the upper part of vagina a polypus about twice the size of a walnut, irregular shape, firm texture; pedicle short and thick; posterior lip of os uteri hypertrophied. A ligature was passed round the pedicle on the 20th; the catamenia recurred on 22nd; the polypus with canula came away on 23rd; pulse rather quick; no other bad symptom. Discharged November 4th.	Recovered.

CASES OF UTERINE POLYPI—*continued.*

	Date.	Name.	Age.	Social condition.	Symptoms and treatment.	Result.
116	Nov. 18th, 1863	Ellen J—,	32	Married; 1 child	Menstruation painful for three months; pain in sacral region; bearing-down pains; yellow vaginal discharge. On the 28th a small polypus was felt; very thin, hardly an inch in length; no pedicle attached to anterior lip of os uteri within cervix. She was kept in bed; the symptoms disappeared, and on December 12th the polypus could just be felt. On the 26th she was discharged.	No operation performed.
117	January 26th, 1864	Elizabeth E—,	41	Married; 3 children; 7 miscarriages	Catamenia profuse and frequent for four years; many miscarriages; profuse watery discharge; a tumour projecting slightly from between the vulva for six months; pain in back; anæmia. No tumour felt through abdomen, internally; a large tumour was found occupying the pelvis, nearly filling it up; it was about the size of a large fœtal head; the finger could be passed readily between it and the vaginal wall all round; at the most dependent part there was a small projection from the main body of the tumour, forming a second tumour; the os uteri could not be felt. On February 9th a ligature was passed round the root of the second tumour; no bad symptoms. On the 11th it came away; on the 16th the ligature was re-applied as high up as the canula could reach and beyond reach of finger; this caused acute pain in left side of uterine region, which lasted for hours; discharge very offensive and profuse. On the 19th the	Recovered.

						canulæ were removed; the ligature was left applied; the lower part of tumour was sloughing; the ligature came away on 21st; discharge very offensive; slight feverish symptoms; great debility. On the 22nd a considerable portion of the sloughing mass was removed with the forceps, and on the 23rd the whole of it was got away. She rapidly recovered, and was discharged March 9th. She has since been heard from, and is quite well and strong.	
118	June 1st, 1864	Jane E—	33	Single; 1 child	Menstruation frequent and profuse for two years; thin yellow discharge; slight bearing-down pains; aching pain in back; anæmia. The whole of the pelvis was filled up by a large tumour, larger than a fœtal head; the finger can be passed between it and the vagina all round as high as one can reach; the os uteri cannot be felt. The surface of the tumour is smooth, soft externally, hard internally; it has no sensation, and is quite immovable. On July 9th a ligature was passed round the tumour as high up as the canulæ would reach. The canulæ were removed on 12th; ligature left behind; pulse quick; no pain or fever; discharge very offensive. On the 13th some ineffectual efforts were made to extract the tumour, and again on the 16th. On the 17th the ligature came away, and the polypus was felt rolling about; with considerable difficulty it was extracted by means of the craniotomy forceps. (The tumour was composed of a number of fibrous tumours, oval form, flattened sides, from one and a half to two inches in long diameter, and one inch in short, connected together by areolar tissue.) She rapidly recovered, and was discharged August 8th. She has not been heard from since.	Recovered.

CASES OF UTERINE POLYPI—*continued.*

	Date.	Name.	Age.	Social condition.	Symptoms and treatment.	Result.
116	Nov. 18th, 1863	Ellen J—,	32	Married; 1 child	Menstruation painful for three months; pain in sacral region; bearing-down pains; yellow vaginal discharge. On the 28th a small polypus was felt; very thin, hardly an inch in length; no pedicle attached to anterior lip of os uteri within cervix. She was kept in bed; the symptoms disappeared, and on December 12th the polypus could just be felt. On the 26th she was discharged.	No operation performed.
117	January 26th, 1864	Elizabeth E—,	41	Married; 3 children; 7 miscarriages	Catamenia profuse and frequent for four years; many miscarriages; profuse watery discharge; a tumour projecting slightly from between the vulva for six months; pain in back; anæmia. No tumour felt through abdomen, internally; a large tumour was found occupying the pelvis, nearly filling it up; it was about the size of a large fœtal head; the finger could be passed readily between it and the vaginal wall all round; at the most dependent part there was a small projection from the main body of the tumour, forming a second tumour; the os uteri could not be felt. On February 9th a ligature was passed round the root of the second tumour; no bad symptoms. On the 11th it came away; on the 16th the ligature was re-applied as high up as the canula could reach and beyond reach of finger; this caused acute pain in left side of uterine region, which lasted for hours; discharge very offensive and profuse. On the 19th the	Recovered.

					canulæ were removed; the ligature was left applied; the lower part of tumour was sloughing; the ligature came away on 21st; discharge very offensive; slight feverish symptoms; great debility. On the 22nd a considerable portion of the sloughing mass was removed with the forceps, and on the 23rd the whole of it was got away. She rapidly recovered, and was discharged March 9th. She has since been heard from, and is quite well and strong.	
118	June 1st, 1864	Jane E—	33	Single; 1 child	Menstruation frequent and profuse for two years; thin yellow discharge; slight bearing-down pains; aching pain in back; anæmia. The whole of the pelvis was filled up by a large tumour, larger than a fœtal head; the finger can be passed between it and the vagina all round as high as one can reach; the os uteri cannot be felt. The surface of the tumour is smooth, soft externally, hard internally; it has no sensation, and is quite immovable. On July 9th a ligature was passed round the tumour as high up as the canulæ would reach. The canulæ were removed on 12th; ligature left behind; pulse quick; no pain or fever; discharge very offensive. On the 13th some ineffectual efforts were made to extract the tumour, and again on the 16th. On the 17th the ligature came away, and the polypus was felt rolling about; with considerable difficulty it was extracted by means of the craniotomy forceps. (The tumour was composed of a number of fibrous tumours, oval form, flattened sides, from one and a half to two inches in long diameter, and one inch in short, connected together by areolar tissue.) She rapidly recovered, and was discharged August 8th. She has not been heard from since.	Recovered.

CASES OF UTERINE POLYPI—*continued.*

	Date.	Name.	Age.	Social condition.	Symptoms and treatment.	Result.
119	May 25th, 1862	Mrs. G—	45	Married; several children	Profuse menstruation during a considerable period; afterwards constant hæmorrhage from the uterus. A polypus the size of a large walnut felt within the os uteri, which was much dilated; no part of the polypus had passed through the os uteri. The hæmorrhage becoming alarming and the polypus not escaping through the os, it was determined to tie it, and the ligature was applied with the double canula without much difficulty. Several days having elapsed before the ligature came away, and the polypus being in a decomposed state, it was resolved to remove the ligature and extract the polypus with the forceps, which was readily done, and the patient recovered in the most favorable manner.	Recovered.
120	June, 1864	Mrs. J—	—	Married; several children	Uterine hæmorrhage; pain in the region of the uterus and rectum, and sense of bearing down; the large polypus in the vagina, which rendered it difficult to reach the os uteri, was supposed by the medical attendant to be the uterus retroverted. The ligature was applied with the double canula, but not without some difficulty; it was tightened daily until the polypus was in a sloughing state, and then it broke. No attempt was made to apply a second ligature; tepid water was injected frequently into the vagina, and in a few days the polypus was extracted with the forceps, and the patient recovered without any unfavorable symptoms.	Recovered.

ON

AMPUTATION OF THE LEG

BY A

LONG RECTANGULAR FLAP FROM THE CALF.

BY

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Received Feb. 10th—Read May 23rd, 1865.

In amputation of the leg below the knee Mr. Hey advocated the plan of making a long flap, from the back of the leg.[1] The large crural nerve in this operation was either left in the flap or had to be dissected out.

Mr. Teale recommends the formation of a long flap from the front of the leg, as forming a covering consisting of parts free from large nerves, and as furnishing the material for a soft cushion movable over the ends of the bones, which enables the patient to bear a part of his weight upon the extremity of the stump. This is a point of great importance where an artificial limb has to be worn, for there can be no doubt, as Mr. Teale has expressed it, that an equal distribution of the pressure between the end of the stump and the upper part of the leg secures the greatest degree of firmness of step.

[1] 'Practical Observations in Surgery,' chap. xxiii.

In the following cases the advantages of a thick flap from the back of the leg, as recommended by Mr. Hey, were combined with the security against any large nerves being left in the extremity of the stump, as in the proceeding recommended by Mr. Teale. In the operation which I perform, the external incisions are made according to Mr. Teale's plan, but the long flap is obtained from the back instead of from the front of the limb, as represented in Fig. 1. Two

Fig. 1.

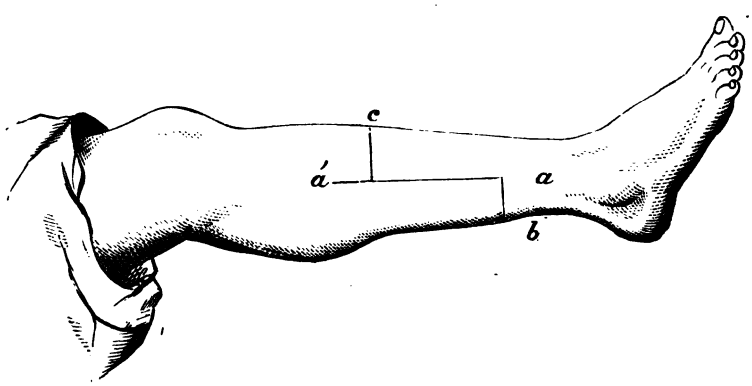

Fig. 1. Showing lines of external incisions.

a—*a'*. Longitudinal incisions.
b. Posterior transverse incisions.
c. Anterior transverse incisions.

Fig. 2.

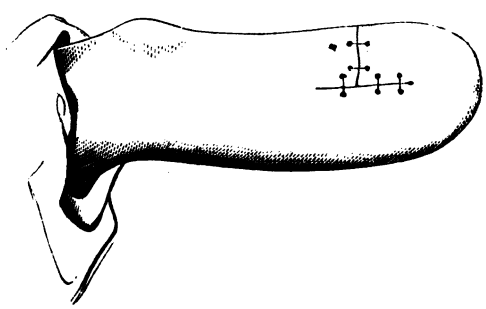

Fig. 2. Showing position of flaps after the operation.

parallel longitudinal incisions are made along the sides of the leg (*a*—*a'*). These are met by a third transverse incision behind (*a*—*b*) which joins the lower extremities of the first two. The incisions thus made form three sides of a square, and extend through the skin and cellular tissue only; a fourth incision (*c*) is made tranversely through the skin in front of the leg, so as to form a flap in this situation one fourth only of the length of the posterior flap. When the skin has become somewhat retracted by its natural elasticity an incision is carried through the parts situated in front of the tibia, interosseous membrane, and fibula. The whole of the parts thus divided are separated close to the periosteum and interosseous membrane, and are reflected upwards to a level with the upper extremities of the first longitudinal incisions.

The deeper structures at the back of the leg are then freely divided in the situation of the lower transverse incision. The conjoined gastrocnemius and soleus muscles are separated from the subjacent parts, and are reflected as high as the anterior flap.

This part of the operation is performed with the greatest facility on account of the loose attachments of these muscles, especially at the lower part of the leg. The deeper layer of muscles, together with the large vessels and nerves, are divided as high as the incisions will permit, and the bones sawn through in the same situation. The flaps are then adjusted as shown in fig. 2.

The long flap thus formed is much thicker than when taken from the front. It is consequently less liable to slough. It affords a much more efficient protection to the ends of the bones, and a thicker and softer pad upon which to rest a part of the weight of the body when an artificial leg is applied.

Case 1.—William F—, æt. 33, was admitted into St. George's Hospital, on the 24th of August, 1863, with extensive disease involving nearly all the tarsal bones of the right foot, and very seriously impairing his general health. On

the 17th of October following the leg was amputated by the operation above described. The patient was at the time very greatly debilitated, and his weakness was still further increased by the occurrence of secondary hæmorrhage after the operation, and on the 9th of November by an attack of laryngitis, for which he was placed under the care of Dr. Barclay. He made, however, an excellent recovery, and was enabled to leave the hospital on the 22nd of November.

A drawing of the stump was made on the 26th of November, 1864, at which time he could walk fourteen miles a day upon his artificial leg without inconvenience. He had now learnt to use his leg so well that a casual observer would not remark that there was anything particular in his gait.

Case 2.—Eliza E—, æt. 21, was admitted into St. George's Hospital, on the 14th of May, 1864, with very extensive disease of the tarsus, for which amputation of the leg was performed, on the 14th of July following, by means of a long rectangular flap from the back.

She was discharged on the 8th of September, with a thick soft cushion moving freely over the extremities of the bones.

On the 10th of December, 1864, the patient was readmitted into the hospital, to have her artificial leg adjusted.

She left the hospital the second time on the 1st of February, 1865, and she could then bear the same amount of pressure upon the end of the stump without inconvenience as upon any other part. In walking she habitually rested about half the weight of her body upon the extremity of the stump, and on the 10th of April she could walk three miles in this way with the greatest ease.

Case 3.—James G—, æt. 39, was admitted into St. George's Hospital, on the 15th September, 1864, both his feet having been run over by a railway train. He had long suffered from disease of the chest, and had lost much blood from the accident before his admission into the hospital. The right leg was removed by a rectangular flap from the back, the left leg by the circular operation.

A drawing of the stump of the right leg was made on the 26th of November, when it was completely healed. This patient was retained in the hospital until the 14th of December for the adjustment of two artificial legs, with which he walked very comfortably after his return home. The right stump contrasted very favorably in this case with the left, which, however, healed about the same time.

Towards the end of January, 1865, during the very cold weather, this patient was seized with a severe attack of bronchitis, which proved fatal, and a preparation shows the condition of the stump at that period.

In two other cases the operation by a rectangular flap from the calf of the leg has been performed; in one of these the patient recovered speedily and well; in the other, which was a secondary amputation (performed after great loss of blood from ulceration of the anterior tibial artery, and under most unfavorable circumstances), the patient died.[1] These cases all fell under my own care, and are, I believe, the only ones in which this particular operation has been performed up to the present time.

[1] In this case there was extensive and old disease of the bronchial glands, which contained a quantity of matter of the appearance and consistence of putty.

AN ACCOUNT OF A CASE

IN WHICH A

SIXPENCE WAS LODGED IN THE LARYNX DURING TEN WEEKS.

BY

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AND

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Received March 15th—Read June 13th, 1865.

On Wednesday, November 2nd, the patient, a muscular man, æt. 27, in good health, was talking with some friends in a public-house, having a sixpence in his mouth, when, something in the conversation exciting his laughter, the sixpence disappeared, and he immediately fell to the ground suffocated.

A neighbouring surgeon was called in, who supposed him to be dying, and at once took him to St. Mary's Hospital, where he arrived about three quarters of an hour after the accident, and was placed in bed on his back. He states that his throat was examined, but that nothing could be detected. In a short time his breathing was easier, and he became tranquil. He remained in the hospital till one o'clock the next day, when he was discharged at his own request. He

affirms that at this time he breathed as well as he had ever done in his life, and could walk fast or go up stairs without any difficulty, but was unable to speak above a whisper. For about a fortnight after the accident he attended at the hospital as an out-patient, complaining of sore throat and pain or difficulty in swallowing liquids, of which symptom he was relieved by treatment. During the succeeding ten weeks his voice remained as before. He lost flesh and strength, but in other respects was in fair health, and never experienced any difficulty of breathing, either on exertion or otherwise.

On the 5th of January his breathing became embarrassed somewhat suddenly, and at 12 o'clock he was brought to see Dr. Sanderson. It appearing, from his statement and from the character of the dyspnœa from which he was suffering, that the sixpence was still in his throat, he was directed to come to the Middlesex Hospital for laryngoscopic examination. On returning home he was suddenly seized in going up to his bedroom with a feeling of suffocation and a fear of approaching dissolution as urgent as he had experienced immediately after the accident. He states that he rushed down stairs in agony, and in doing so tripped. Thereupon the dyspnœa suddenly ceased, and he was again able to breathe as freely as ever.

Next day, at 2.30 p.m., he presented himself at the hospital, when the breathing appeared to be only slightly embarrassed. The sixpence was seen without difficulty on the first introduction of the laryngeal mirror. It was horizontally placed in the glottis, below the false vocal cords, which covered a portion of its circumference at each side, leaving exposed a triangular metallic surface, the base of which was formed by the free edge of the coin, while the apex was overhung by the epiglottis. Between the free edge and the arytenoid cartilages was a transversely oblong breathing space, apparently not wider than a tenth of an inch from front to back, and bounded laterally by the posterior extremities of the false vocal cords and the aryteno-epiglottidean folds. The entire visible laryngeal mucous membrane was red and puffy. His breathing was slow (thirteen re-

spirations per minute), and on listening to his larynx it was found that the two respiratory acts were of nearly equal duration, expiration being slightly longer than inspiration. On percussion it was found that the chest was everywhere resonant, but that the pulmonary margin scarcely extended posteriorly below the level of the ninth dorsal spine in tranquil breathing. On auscultation a feeble inspiratory and expiratory sound of a somewhat tubular character was heard all over the chest, but the natural vesicular murmur could not be distinguished.

It was at once determined to attempt the extraction of the coin with the aid of the laryngoscope, through the upper opening of the larynx; and inasmuch as its horizontal position rendered it impossible to seize it with forceps, bent loops of wire were prepared for the purpose of jerking it out. As a preliminary tentative measure, the patient was held with his head downwards, smartly slapped on the back of his neck, and directed to cough, which he was unable to do from inability to fill his chest. These measures effected a slight apparent alteration in the position of the coin, but were unsuccessful, and, the mucous membrane having become much more red and turgid, resembling crimson plush, it was resolved to have recourse to laryngo-tracheotomy.

After a delay of four hours, in order to gain the consent of the patient's wife, he was put under chloroform, and a cut one inch and a half long was made in the middle line from the thyroid cartilage downwards. The crico-thyroid membrane, the cricoid cartilage, and two or three upper trachial rings, having been divided, the wound was held apart above and below with two pairs of Trousseau's dilators, while search was made for the coin with a forceps introduced from below. It could be felt, but not seized. An attempt to pass one blade of the forceps in the angle of the thyroid cartilage over the anterior edge of the coin was unsuccessful, though the instrument easily slipped through the glottis behind the coin, and could be felt with the forefinger of the other hand in the fauces. In this attempt the coin was displaced upwards within the reach of the finger, by which it was drawn

forwards over the epiglottis on to the root of the tongue. At this moment the patient made a sudden gulp, the coin slipped away, and the patient, who had become conscious, made signs that he had swallowed it. Immediately after, he breathed freely through the glottis, the wound remaining open, and on auscultation over the front of the chest air was heard to enter freely. He was at once placed in bed, in a warm, moist atmosphere. The wound was covered with a sheet of cotton wool.

It is unnecessary to give a detailed report from this time. On the following morning he passed the coin per anum; his breathing was easy and noiseless, sixteen respirations per minute, and his pulse was 84.

On the fourth day his respirations were thirteen, and his pulse 82 per minute.

On the eleventh day his chest was resonant as low as the twelfth dorsal spine, and the vesicular murmur, still singularly weak, could be everywhere heard; his respirations had risen to eighteen, and his pulse had fallen to 64 beats per minute.

For about a week after the operation a little air bubbled through the wound on coughing. Laryngoscopic examinations from time to time revealed a small group of granulations just above the base of the right arytenoid cartilage, near the attachment of the false cord, and diminished puffiness and redness of the laryngeal mucous membrane.

On the 18th of January, when he was made an out-patient, the wound had nearly cicatrized.

By the 20th of February the patient's wound had regained its natural character, but he himself stated that it was not so powerful as it had been before the accident. On examining the larynx it was found that its mucous membrane and that of the epiglottis were still unnaturally red and turgid. The pulmonary signs had undergone no change since he left the hospital. The breathing was still remarkably slow (not more than thirteen respirations per minute), and in many parts of the chest the respiratory murmur was so feeble that it could not be distinguished.

ON A CASE

OF THE

"HÆMORRHAGIC DIATHESIS."

BY

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Received March 24th—Read June 13th, 1865.

THE pathology of the disease, or, to speak more correctly, perhaps, the diseases included in the term hæmorrhagic diathesis, is so obscure, and their occurrence, at least in this country, comparatively so rare, that a single case seems worth recording, if it were only as an addition to the literature of the subject; but the case which I am about to describe is, in many respects, so peculiar, even apart from its being an instance of this kind, that I hope it may be thought worthy of being brought under the notice of the Medical and Chirurgical Society.

For the opportunity of seeing and recording the case I am indebted to the kindness of Mr. Paget and Dr. Allan.

The subject of the disease was a tradesman, æt. 23, who, until 1862, when he left the country and began to live in London, was strong and healthy. Since childhood he had

been subject to occasional vomiting and other symptoms of what was called biliousness, but these never troubled him much, and he was able to do a hard day's work without difficulty. About a fortnight after coming to town he was exposed, during one night, to much cold, and to this exposure the beginning of his illness was attributed, for he was laid up for a month or six weeks afterwards with symptoms of what was supposed, at the time, to be some affection of the kidneys—albuminous and perhaps bloody urine, vomiting, &c. He remained ill for some time, and, as little or no improvement took place in town, he went into the country. Here he soon recovered his usual health, or nearly so, but very soon after his return to town (about the beginning of 1863) he again fell ill, vomiting, purging, and hæmaturia being the most prominent symptoms.

At about the same time a small swelling, like a little boil or acne pimple, appeared on the cheek, near the angle of the mouth, and, after being accidentally rubbed, bled profusely at short intervals for a fortnight, and it was thought that he must have lost altogether two quarts of blood in this time.

The bleeding, however, ceased at last, and did not occur again for nearly a twelvemonth. The vomiting and purging continued to occur very frequently. The matters rejected from the stomach were not peculiar, unless in their sometimes containing a little blood; the fæces were pale; the urine was dark, probably from its containing blood, but no regular examination of it was recorded at this time. During the whole of 1863 the patient's health was much broken by these various disorders, although he was not completely laid up.

In November, 1863 (nearly a year after the first severe hæmorrhage), another small pimple, which had recently appeared on the face, began to bleed with great violence, the blood spurting out forcibly to some distance, and its flow being almost uncontrollable, except by firm pressure. On this occasion the hæmorrhage continued until a common-sized washhand basin was nearly full of blood, notwithstanding the efforts that were made to stop it by pressure, by

powdered matico, &c., and, at last, by the actual cautery. He fainted for a short time during the hæmorrhage.

After some hours the flow of blood ceased, but on the following day it began again, and continued until a considerable quantity, though less than on the last occasion, had been lost. Similar hæmorrhages to these took place, at intervals, for about seven weeks. Sometimes, and most frequently, more or less blood was lost every day, the quantity varying from an ounce or two to half a pint; at other times one or more days passed without any hæmorrhage at all. The longest interval, however, with complete absence of bleeding, during the seven weeks, was only four days.

The blood, apparently arterial, usually spurted out quite suddenly from one of the pimples on the face, and often with a jet to the distance of a foot or more from the patient. It was always difficult, and sometimes almost impossible, to stop the bleeding, on account of the force with which the blood flowed. Pressure was the most effectual means, but even with this the blood welled out from beneath unless the pressure were very firm. Astringents did only partial good, and often none at all until a considerable quantity of blood had been lost.

The bleeding was chiefly, but not entirely, from the pimples on the face. Sometimes the nose bled, but less violently than the pimples. In small quantities, too, blood was occasionally present in the fæces and in the matters rejected by vomiting. It was almost constantly present in the urine.

The pimples were not unlike those of common acne, but somewhat flatter and of a duskier colour, and with a small areola of the same tint, or darker. Only one was larger, and that not more than double or thrice the size, though the dark purplish areola made it look bigger. There were about five or six on the face and two or three on the neck, but they did not all of them bleed. The first pimple, which was mentioned as bleeding in January, 1863, still remained, and was, therefore, of about a year's duration; the others had appeared subsequently, at different times during the patient's

illness. There were no similar pimples on any other part of the surface of the body, but several small, dark red points, scarcely raised, and like very minute purpura spots, appeared on the lower part of the legs.[1]

The blood was said to coagulate always more or less firmly. A specimen that I saw, and examined carefully, looked in every respect like ordinary blood, and had separated, in the usual manner, into clot and serum; and unless that the clot was rather soft, it appeared both to the naked eye and under the microscope like that of ordinary blood.

During the seven or eight weeks that the bleedings continued, the patient became thinner and weaker, and was, at last, almost entirely confined to his bed. But he did not appear, even at this time, nearly so exhausted as might have been expected from the large quantities of blood that he had lost; the conjunctiva was red, and indeed looked rather congested; his face was not very pale; it wore a dull and heavy, almost stupid, expression, and was said to be much more dull, or even bloated, immediately before an outbreak of the hæmorrhage. The complexion was muddy rather than anæmic. He was quite rational, but answered questions in a hesitating manner, as if he only slowly comprehended them; he slept badly, and was sometimes "light-headed;" the pulse (112) was full, irritable and jerking; the bowels were rather costive, although previously, for many months, as before mentioned, they had acted too frequently. The urine was dark, dim red, and turbid, with a rather copious sediment of blood and lithic acid, and a little oxalate of lime; the heart and lungs seemed quite healthy, and no anæmic murmur could be heard at the base of the former. The abdomen, too, was quite normal, and there was no apparent enlargement of any of its viscera.

Nothing at all definite could be assigned as a cause of his illness. He was always of very sober habits, and had been married for three years. The house in which he lived was of

[1] They were quite small enough to escape observation unless looked for, but, after careful inquiry, I came to the conclusion that they did not appear until some time after the commencement of the hæmorrhages.

good size, though dark and close, with stables adjoining the premises behind. Other people, however, who lived in the house, were not affected with any special or like complaint, and his predecessor had lived there for many years, without any particular disease in himself or family.

Until within the date of his present illness the patient had never noticed that slight wounds caused any large or long-continued hæmorrhage, although he thought he was always, as he expressed it, "inclined to bleed." A short time, however, before the commencement of the severe hæmorrhages, he grazed one of his knuckles, and blood oozed from the injured part, though not in any large quantity, for two or three days. None of his family were specially subject to hæmorrhage, with the exception of his mother, who had often suffered from very severe epistaxis.

With regard to the treatment that was adopted in this case, it may suffice to say that it was without any other than temporary good effect either on the hæmorrhages or the patient's general health. Various means were tried, but without avail, and it was considered best to remove the patient to the country, as no benefit took place in town. He was accordingly removed about the beginning of January, 1864, nearly two months after the recurrence of the more alarming hæmorrhages. During the removal he seemed much exhausted and was partially insensible.

For an account of his progress during the next few weeks I am indebted to the kindness of Dr. Mott.

Some bleeding from the nose occurred for a short time, soon after his arrival in the country, and, two days afterwards, a small quantity of blood escaped from one of the pimples on the face. On the same day, and for the first time, he had two somewhat violent epileptic fits, and during the following night was sleepless and delirious, and required restraint to keep him in bed.

These were, however, the last serious symptoms that he had. On the day following his general health began to improve, at the same time that the urine became clearer, and the albumen began to disappear. No more bleeding occurred

from any part of the body, excepting a small quantity of blood which was passed with the fæces on one or two occasions, and from this time onward he speedily regained health and strength. Oddly enough, however, he lost all remembrance of the few days immediately preceding and following his removal from town, and could not recognise anybody whom he had seen for the first time during that period, although, at the time, he had conversed with them quite rationally.

After he had been in the country for about two months I again saw him. He was looking rather pale, perhaps paler than during his illness, but seemed nearly well, and said he felt so. The urine was clear and healthy, and with a bare trace of albumen.

The pimples on the face remained, and were scarcely at all altered. One of them, the largest, which had been touched, during a violent hæmorrhage, with the actual cautery, had still a purplish areola about a quarter of an inch wide.

Soon after this time he returned to his house in London, and in two or three weeks afterwards, as if from the change, and possibly, also, from trouble in business matters, he again seemed to be falling into a bad state of health. The chief symptoms were general debility and inaptitude for exertion; dulness of countenance and intellect; furred tongue; irritable pulse, &c.; the bowels were sometimes relaxed, and he had frequently passed small quantities of blood with the fæces. The urine, however, remained clear, and he had no return of hæmorrhage from any other part than the rectum. The pimples on the face remained almost as they were, or had, perhaps, become a little smaller, and there were no purpura-like spots on the legs or elsewhere.

December, 1864.—At this date (about eight months after the last note), he was well, and had had no return of hæmorrhage. He had been living during almost all the time in the country.

Although it would be improper to make any general deductions from a single case, like the present, it may be well to direct attention, very briefly, to its more important features,

and see what likeness, if any, can be discovered to other recorded instances of the disease, or to any other diseases, not usually supposed to be caused by the same hæmorrhagic diathesis.

And, in the first place, may be noticed the marked illustration afforded by the present case of the differences between the idiopathic and what may be called the simply traumatic varieties of the disease. The congestion which often preceded and which was only partly relieved by the hæmorrhage, the occasional delirium, the epileptic fits, and the absence of those symptoms of anæmia which follow a mere loss of blood, over and above the spontaneous occurrence of the bleedings, point to some condition of the system far beyond what can be accounted for by a want of coagulating power in the blood, or thinness of the coats of the blood-vessels, such as have been observed in cases of fatal bleeding after slight wounds.[1] It is not necessary, however, to do more than refer to these differences, inasmuch as those symptoms which best illustrate them are not peculiar to the present case.

In the interesting memoir of this disease by Grandidier,[2] cases are related which, in many respects, resemble this one, especially in the spontaneous and intermittent character of the bleedings, and in the signs of general congestion which preceded the hæmorrhages, as well as in the immunity from danger after slight wounds, in the intervals.

There is a reference, moreover, in Andral's 'Pathological Anatomy,' to two somewhat similar cases. "Two children, the subjects of this disease, presented, at various intervals, on their legs and thighs, a number of blue spots which swell to the size of a pigeon's egg and then assume a yellowish-green shade; they do not bleed unless when opened artificially, but the hæmorrhage continues until the patient falls into a swoon, and the body becomes pale as death. The further progress of the bleeding is arrested by the pressure of the

[1] 'Trans. of Med. and Chir. Soc.,' vol. viii, p. 227. Cooper's 'Surg. Dict.,' edited by Lane, vol. i, p. 864.

[2] 'Die Hæmophilie oder die Bluterkrankheit,' 1855.

finger on the orifice, which it is generally necessary to continue for twenty-four hours." The cases, however, have a greater likeness to some which are quoted by Grandidier than to the present, especially in the large size and other characters of the swellings.

"Redman Coxe mentions a case in which, without any wound, blood sweated from the ears, the nose, and the skin of the occiput; later, also, at the shoulders, the belly, the tips of the fingers, and toes," &c.[1]

Other cases more or less like these might be referred to, but I can find none which more closely resemble the present; and there remain, therefore, a few points in the latter which are peculiar, and worth noticing, although, as before remarked, no general deduction can be safely made from them. The force with which the blood flowed, the large quantities which escaped, the delirium, convulsions, &c., are such as happen occasionally in other instances of hæmorrhage depending on this special diathesis, and are not peculiar to the present case.[2]

1. The pimples on the face, from which the blood flowed so profusely, had some peculiar characters. I do not remember seeing, previously, any exactly resembling them, but they were, most of them, sufficiently like acne pimples to make it possible that they may have been some eruption allied to this, whose characters were masked by the blood disease. Whether this were so, or whether they were a diminutive species of the "blood-swellings" sometimes observed in this disease, and described by Grandidier and others, I am unable to say.

2. The purpura-like specks on the legs were small and indistinct, and scarcely marked enough for one to decide positively on their nature, but they are interesting in connection with the likeness of the present disease to purpura, the so-called "eruption" of which they closely resembled.[3]

The absence, however, of a well-marked eruption, and the

[1] Virchow, 'Handbuck der Speciellen Path. und Therap.,' Band i, p. 265.
[2] Ibid., p. 267.
[3] Petechiæ are, indeed, described by Grandidier and others as often

presence of the other peculiarities preceding and accompanying the hæmorrhages, prevent more than a passing notice of the likeness (often referred to by authors) between the two diseases. It is worthy of remark, however, that Grandidier, after describing the differences between them, and giving his opinion that they are essentially distinct, admits that there are some transition-forms, as it were, in which the diagnosis is very difficult.

3. The decided benefit which seemed to be almost immediately derived from change of air, certainly deserves special consideration, and more particularly as it occurred more than once, and after other means had been long tried in vain. A third time, too, the patient appeared to be on the eve of a fresh attack of the disease, after only a short residence in town. Mr. Moore remarks "that the characteristics of the hæmorrhagic diathesis may be induced in a young and previously healthy person by the privation of fresh air, light, and nourishing food, and by confinement in a damp low situation, and they will disappear again on placing the patient in conditions favorable to health."[1] In the present case, however, the cause of the disease could not be traced definitely to any special defect in the hygienic condition of the house or district in which the patient lived. But it may be that one, in whom there exists a tendency to such disease, may have the predisposition excited to action by particular and quite occult hygienic influences; and that the only means of freedom may be in the careful avoidance of all such as are found by experience to be hurtful. This seems, at least, a fair enough inference from the history of the present case, and among the records of some other diseases an analogy is not altogether wanting.

accompanying the hæmorrhagic diathesis, but they appear to be usually of considerable size. See Grandidier, l. c., Wachsmuth, 'Die Bluterkrankheit,' 1849.

[1] Holmes's 'System of Surgery,' vol. i, p. 656.

A SECOND SERIES

OF

FIFTY CASES OF OVARIOTOMY;

WITH

REMARKS ON THE SELECTION OF CASES FOR THE OPERATION.

BY

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In the forty-sixth volume of the 'Transactions' of this Society, a paper is published 'On the History and Progress of Ovariotomy in Great Britain,' illustrated by a series of fifty cases in which I had completed this operation, and of six other cases in which I had commenced the operation but had not completed it, or had made an exploratory incision in aid of diagnosis. The same volume of the 'Transactions' contains the report of another case where I performed the operation for the second time upon the same patient.

The only conclusion which I endeavoured to impress upon the Society from these cases was, that ovariotomy is an operation which can no longer be regarded as unjustifiable; but that, on the contrary, it becomes the clear duty of the sur-

geon to perform it in certain cases. I did not attempt to draw any line between cases which are favorable or unfavorable for operation, nor to point out the conditions by which he may approximatively estimate the probability of success or of failure in any given case. I felt that a larger experience was necessary before this could be done. But now that opposition to the principle of the operation has almost ceased, and it is recognised almost universally in this country and in America as a lawful or "legitimate" operation, and the influence of British example is extending to France and Germany, it appears that a second series of fifty cases, forming, with the first series, one hundred cases in which I have completed the operation up to June, 1864, may furnish a sufficient number of facts upon which the inquiry may be begun—although it can be little more than begun—as to the selection of cases for the operation; the rejection of those in which there can be no reasonable hope of recovery; the conditions in which hope and fear are so equally balanced that a patient and her friends may fairly be called into consultation; and those which are so favorable that the operation may properly be recommended to a patient as one of moderate or small risk compared with the nature and progressive danger of the disease.

The following table contains some particulars as to the second series of fifty cases in which ovariotomy was completed; of one case in which it was commenced but not completed, and of two cases in which an exploratory incision was made.

Table of Cases to accompany Mr. Spencer Wells's second series of Fifty Cases of Ovariotomy.

SERIES I.—Cases in which Ovariotomy was completed—50 cases; 33 recoveries; 17 deaths.
„ II. „ „ was commenced, but not completed—1 case. 1 death.
„ III. „ an exploratory incision was made—2 cases. Both recovered.

SERIES I.—*Cases in which Ovariotomy was completed.*

No.	Date.	Age.	Condition.	History, &c.	Result.
51	1862. Dec.	42	Married	Albuminuria; firm adhesions; a pailful of fluid	Recovered.
52	Dec.	53	Single	Non-adherent adenoid tumour, surrounded by ascitic fluid	Recovered.
53	1863. Jan.	34	Married	Large cyst containing 72 pints of fluid.	Recovered. Child born fifteen months after operation.
54	Feb.	25	Single	26 lbs. of fluid; 4 lbs. of adenoma; firm adhesions; short pedicle	Died in 44 hours.
55	Feb.	56	Married	Tapped thrice; large cyst, 69 pints of fluid; firm adhesions	Recovered.
56	March	36	Single	Large, adherent, solid mass, and 36 lbs. of fluid	Recovered.
57	March	29	Single	46 lbs. of fluid and 4 lbs. of small cysts, with hair, fat, and teeth; adhesions	Recovered.
58	March	36	Single	13 lbs. of fluid, 2 lbs. of fat; two hair cysts; no adhesions	Recovered.
59	March	26	Single	About 30 lbs.; no adhesions	Died, 27 days after, of pyæmia.
60	March	26	Married	Ascitic fluid surrounding small dendritic growths	Died in 54 hours.
61	April	61	Married	Large cyst; firm adhesions	Recovered. Died 4 months after.
62	April	19	Single	15 lbs. of fluid and 1 lb. of cyst wall; no adhesions	Recovered.
63	April	37	Married	33 lbs. of fluid and 14 lbs. of adenoma; adhesions, parietal, omental, and intestinal	Recovered. Died 3 months after.

No.	Date.	Age.	Condition.	History, &c.	Result.
64	May	33	Married	One sac containing 20 lbs. of fluid, and a mass of small cysts; no adhesions	Recovered.
65	June	50	Married	36 lbs. of fluid; extensive parietal and omental adhesions	Died, 54 hours after, of septicæmia.
66	June	44	Married	34 lbs. of fluid; 3 or 4 lbs. of solid; parietal adhesions	Recovered.
67	June	37	Married	Multilocular cyst; glairy contents; adhesions; rupture of cyst	Died 80 hours after.
68	June	29	Single	Multilocular cyst; no adhesions	Recovered.
69	June	55	Single	Non-adherent simple cyst	Recovered.
70	June	49	Married	Multilocular cyst; adhesions to abdominal walls and intestines	Recovered.
71	July	35	Single	No adhesions; 21 lbs. of fluid	Died in 78 hours; clot in heart.
72	July	35	Single	Multilocular cyst; fibroid outgrowth from uterus also removed	Died in 44 hours.
73	Aug.	55	Single	Simple cyst; no adhesions	Recovered.
74	Aug.	24	Single	Multilocular cyst; no adhesions	Died, in 82 hours, of peritonitis.
75	Sept.	35	Single	Multilocular cyst; no adhesions; 15 lbs. fluid, and 1 lb. adenoid	Died, in 40 hours, of peritonitis.
76	Oct.	35	Married	Six tappings; ascites; rupture of large multilocular cyst	Recovered.
77	Oct.	23	Single	Multilocular; firm adhesions	Recovered.
78	Nov.	33	Single	Multilocular; extensive adhesions	Died, on eighth day, of septicæmia.
79	Nov.	32	Single	Non-adherent; 17 pints; pedicle returned	Recovered.
80	Nov.	19	Single	One cyst, non-adherent, the size of an adult head, and a cluster of small cysts as large as an orange	Recovered.
81	Nov.	33	Widow	Ascites and prolapsus uteri; 43 lbs. of fluid; collapsed cyst, size of an adult head	Died, on eighth day, of hæmatocele.

82		Dec.	57	Married	Ascites; multilocular tumour larger than an adult head; adhesions	Died, on third day, of peritonitis.
83		Dec.	36	Married	Both ovaries removed; A, multilocular, size of melon; B, simple, size of adult head	Died, on third day, of peritonitis.
84	1864.	Feb.	23	Single	No adhesions; simple cyst; 16 lbs. of fluid	Recovered.
85		Feb.	41	Married	Adherent multilocular tumour, weighing 7 lbs. 3½ oz.; ascites; prolapsus uteri	Recovered.
86		March	57	Married	Adherent multilocular tumour, weighing about 40 lbs.	Recovered.
87		March	23	Single	Multilocular, non-adherent; one third larger than the adult head	Recovered.
88		March	43	Single	Multilocular, 10 to 15 lbs.; extensive adhesions	Recovered.
89		April	23	Single	Non-adherent, simple cyst, twice the size of a fœtal head; studded with tubercle	Died 114 hours after.
90		April	32	Married	Non-adherent cyst; 12 pints of fluid and 2 lbs. adenoma	Recovered.
91		April	45	Married	Cysto-sarcomatous tumour, extensively adherent, weighing 59 lbs.	Recovered.
92		April	50	Married	26 pints of fluid; adherent mass, weighing 7½ lbs.	Died in 64 hours.
93		April	21	Single	Non-adherent simple cyst of broad ligament; pedicle returned	Recovered.
94		April	40	Widow	Multilocular adherent cyst, size of adult head; 18 pints of fluid	Recovered.
95		May	47	Married	Multilocular cyst; no adhesions; pedicle returned	Recovered.
96		May	46	Single	Ruptured pseudo-colloid; 17 lbs. of jelly, 6 lbs. of tumour	Died in 44 hours.
97		May	40	Single	Multilocular; parietal and intestinal adhesions; 7 quarts of fluid full of altered blood	Died in 67 hours.
98		May	33	Married	Multilocular; no adhesions	Recovered.
99		May	23	Single	Multilocular, weighing 2 lbs. 6 oz.; 26 pints of fluid; extensive adhesions	Recovered.
100		June	54	Married	Both ovaries removed, each the size of an adult head; no adhesions	Recovered.

SERIES II.—*Case in which Ovariotomy was commenced, but not completed.*

No.	Date.	Age.	Condition.	History, &c.	Result.
1	1864. Jan.	33	Married	Adherent multilocular tumour; not completely removed	Died in 23 hours.

SERIES III.—*Cases in which an Exploratory Incision was made.*

No.	Date.	Age.	Condition.	History, &c.	Result.
1	1862. Dec.	22	Single	Fluid of tubercular peritonitis evacuated by small incision	Recovered. Is now in good health.
2	1864. May	39	Married	Cyst ruptured spontaneously; ovarian fluid removed from peritoneal cavity by small incision	Recovered. Has been tapped several times since.

Of the 100 patients upon whom the operation was completed, 66 recovered and 34 died, and it is curious that in both the first and second series of 50 cases the results are precisely the same, namely, 33 recoveries, and 17 deaths. But instead of six cases of incomplete or exploratory operations which accompanied the first series, only three accompany this second series. It would be unfair to regard these cases as instances of mistaken or even of imperfect diagnosis; because, in nearly all of them the actual result was anticipated before the incision was made, and it was made rather to avoid possible error, or yielding to the urgent solicitation of dying women, than with any confident hope of success. Still, it is satisfactory that increasing experience has lessened the proportion of these incompleted cases, and that while 6—or 12 per cent.—occurred in the first series of fifty, only 3—or 6 per cent.—occurred in this second series of fifty—or 9 per cent. on both series together.

Age.

In order to examine the influence of the *age* of a patient upon the result of ovariotomy, I have prepared the following table, which shows the ages of the first 100 patients upon whom this operation was completed, with the result:

Ages.	Cases.	Recoveries.	Deaths.	Mortality per cent.
15 to 20	4	4	0	0
20 to 25	13	11	2	15·38
25 to 30	14	8	6	42·85
30 to 35	15	7	8	53·33
35 to 40	15	8	7	46·66
40 to 45	13	10	3	23·07
45 to 50	8	5	3	37·50
50 to 55	9	6	3	33·33
55 to 60	8	6	2	25·00
60 to 65	1	1	0	0
	100	66	34	

The small mortality shown in patients below the age of

twenty-five and above the age of forty, and the comparatively high mortality between those ages, is remarkable.

CONJUGAL CONDITION.

Of these 100 patients, there were—

		Recovered.	Died.	Mortality per cent.
Married .	47	31	16	34·04
Unmarried .	53	35	18	33·96
	100	66	34	34·00

This shows that the mortality was nearly equal among married and unmarried women *at all ages*. The numbers are far too small to enable us to ascertain the comparative mortality *at any age* between the married and unmarried.

SOCIAL CONDITION.

I need not say that the results of operations in hospital and private practice are affected by many other causes besides the social condition of the patients; but it may be of some value in ascertaining the effect of modes and habits of life of patients upon the mortality of ovariotomy to state that of the 100 cases there were—

		Recovered.	Died.	Mortality per cent.
Hospital cases	54	38	16	29·6
Private cases	46	28	18	39·1
	100	66	34	

INFLUENCE OF SEASON.

On the whole, the mortality was rather lower in the spring and summer than in the autumn and winter months. It was highest in December and January, but it was lowest in November and March. Hence, looking to the small dif-

ference between the mean temperatures of November and December—the months of lowest and highest mortality, or between January and March—the months of the next highest and lowest mortality, it seems probable that the result of ovariotomy is not much influenced by the season when it is performed.

SIZE AND CONNECTIONS OF THE TUMOURS.

Adhesions.—In 41 cases there were no adhesions, or they were so slight as to be almost unnoticed; of these patients 27 recovered and 14 died, the mortality being 34¼ per cent. In 59 cases, adhesions were very extensive; of these patients 39 recovered and 20 died—a mortality of 34 per cent. So that, as regards adhesions to the abdominal wall, omentum, or intestines, it would appear that they have little, if any, influence upon the mortality, and that the importance which has been attached to the diagnosis of adhesions before operation has been very greatly and unnecessarily exaggerated. But the diagnosis of adhesions within the pelvis is of very great importance, as the attachments to the bladder or rectum may be almost inseparable without great and immediate danger to life. The same may be said of attachments around the brim of the pelvis, the separation of which would endanger the iliac vessels or the ureters. And the closeness of the connection between the uterus and the ovarian tumour —in other words, the length of the pedicle—becomes of great importance, as upon the length depends the possibility of keeping the end of the secured pedicle outside the peritoneal cavity or the necessity for leaving it within this cavity. I do not propose, in the present paper, to enter upon the question of diagnosis; but as it is seldom difficult to ascertain, before operation, the probable extent of pelvic adhesions, and the probability of being able to carry out the extra-peritoneal treatment of the pedicle, the following facts appear to be important.

1. Of the 100 cases, the pedicle was secured and kept outside the peritoneal cavity by a clamp in 66 patients. The

result was 48 recoveries and 18 deaths, or a mortality of 27·3 per cent.

2. In 16 cases the pedicle was secured by a ligature, but was also kept outside. The result was 10 recoveries and 6 deaths—a mortality of 37·5 per cent.

Taking these two series of cases together, we have 82 cases in which the end of the pedicle was kept outside the abdominal cavity, the result being 58 recoveries to 24 deaths—or a mortality of 29·3 per cent.

3. In 5 cases the pedicle was tied and allowed to sink into the abdomen, one or both ends of the ligature being brought out between the edges of the wound. Of these patients, 4 died and 1 recovered—a mortality of 80 per cent.

4. In 3 cases the clamp was used at the operation, but was taken away after a ligature had been applied behind it, and the pedicle was allowed to sink into the abdomen, the ends of the ligature being kept between the edges of the wound. 1 of these patients recovered and 2 died—a mortality of 66 per cent.

Taking these two series of cases together, we have 8 cases in which the end of the pedicle was left within the abdomen, the ends of the ligature being kept between the lips of the wound; the result being 6 deaths and 2 recoveries—a mortality of 75 per cent.

5. In 9 cases the pedicle was tied, the ends of the ligature were cut off short, and were allowed to sink into the abdomen with the end of the pedicle, the wound being closed. The result was 5 recoveries and 4 deaths—a mortality of 44 per cent.

6. In one successful case the *écraseur* was used.

The practical inference from these facts, so far as regards the selection of cases for ovariotomy, is that the probability of success is nearly doubled where the attachments of an ovarian tumour permit the end of the secured pedicle to be kept outside the cavity of the abdomen until it has separated.

The *size* of an ovarian tumour has not, by itself, appeared to affect the result; but size and solidity together, by affect-

ing the length of the incision necessary for the removal, appear to be of some importance. If there be but little solid or semi-solid substance present—which is generally easily discovered before operation—large adherent cysts holding fifty, sixty, or seventy pounds of fluid may be removed after the contents of the cyst have been evacuated through an opening only just large enough to admit one of the operator's hands. The result of such cases has been very satisfactory; but the mortality has been greater when longer incisions have been necessary. The number of inches is a very imperfect mode of judging of the length of incision in these cases; for in a small woman with a tumour of moderate size, an incision of eight or ten inches would extend almost from sternum to pubes; while in a large woman, whose abdomen is greatly distended by a large cyst, an incision of this length may be made below the umbilicus, and after the contraction of the abdominal wall, the cicatrix may not be more than three or four inches long, so that, in selecting a case for operation, it becomes important to judge whether a cyst or tumour can probably be removed by an incision which does not extend above the umbilicus. My impression is, that when this can be done, the probability of success is much greater than when it becomes necessary to extend the incision much above the umbilicus. Of the 100 cases, it was necessary to carry the incision above the umbilicus in twenty patients. Of these nine died—a mortality of 45 per cent. In 80 cases the incision did not extend above the umbilicus. Of these patients 25 died—a mortality of 31 per cent., or 14 per cent. in favour of the shorter incisions.

It was my intention to have prepared some tables showing how far the age and condition of the patient, the size of the tumour, the existence of adhesions, the length of the pedicle, and any other particulars which can be ascertained or made out with tolerable accuracy when the question of operation is discussed, have affected the result in the 100 cases upon which this paper is founded. But the numbers are too small to give any value to such calculations where the conditions are so complex. I think, however, that the experience has

been sufficient to warrant the acceptance of some such rule as the following:

"The probable result of ovariotomy can be estimated with far greater accuracy by a knowledge of the general condition of the patient, than by the size and condition of the tumour."

In other words a large tumour, extensively adherent, in a patient whose heart and lungs, and digestive and eliminative organs are healthy, and whose mind is well regulated, may be removed with a far greater probability of success, than a small unattached cyst from a patient who is anæmic or leukhæmic, whose heart is feeble, whose assimilation and elimination are imperfect, or whose mind is too readily acted upon by either exciting or depressing causes. I believe this to be the explanation of the facts which have led some superficial observers to assert that the more advanced the disease the greater, and the earlier the stage of the disease the less, is the probability of recovery. I am convinced that this reasoning is based on the observation of a few exceptional cases where small unattached tumours have been removed with a fatal result from unhealthy persons; or where large attached tumours have been successfully removed from persons who have otherwise been constitutionally sound. In this question, as in every other where we have to decide upon matters of life and death, medicine and surgery in their widest sense must cordially co-operate in the service of humanity.

ON THE OBSTACLES TO THE RE-ESTABLISHMENT OF NATURAL RESPIRATION AFTER THE PERFORMANCE OF TRACHEOTOMY.

CASES WITH REMARKS.

BY

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The hindrances to the withdrawal of the canula and to the restoration of the passage of the larynx, to which I wish to draw attention, are such as only take effect after the performance of tracheotomy, and as a consequence of that operation. They have no reference to the persistence of any cause of obstruction that may have necessitated the introduction of the tracheal tube.

Case 1.—George S—, a delicate-looking, pallid boy, came into the Children's Hospital, April 4th, 1864. He had suffered from symptoms of laryngeal obstruction for some years, ever since falling into a canal and taking cold.

During the day-time his breathing was difficult and stridulous, and occasionally at night he was seized with a violent paroxysm of dyspnœa; which more than once put his life in great peril. At Dr. West's request, a fortnight after the child's admission I opened the trachea just below the cricoid cartilage. The operation gave immediate and permanent relief; but from the date of its performance the patient was never able either to wear a cork in the orifice of the canula, or to bear the removal of the tube for more than a few seconds.

The ability to inspire through the larynx was entirely lost, though on a momentary closure of the canula he could blow a whistle, and even speak in a harsh stridulous voice. As the child seemed to be gradually losing even this diminished expiratory power, about a fortnight after the operation he was put under chloroform (which was easily administered through the tracheal canula), and the finger being introduced into the throat, an examination was made. The epiglottis and aryteno-epiglottidean folds were found much thickened, indurated, and corrugated, so as satisfactorily to account for the dyspnœa under which he laboured previous to the performance of tracheotomy, but the condition was not sufficient to account for the complete inability to inspire from which he now suffered.

On examining the larynx from below, through the wound in the trachea, it was found that abundant vascular granulations had collected above and around the upper end of the canula, so as almost completely to block up the entrance to the larynx from below.

By the introduction of minute tents formed of the "Laminaria digitata," or sea-tangle weed, the cavity of the air-tube above the canula was soon cleared of granulations, and its normal calibre restored; other means were also taken to remedy the chronic thickening about the upper part of the larynx. At the present date, however, the boy remains unrelieved; he still wears a canula in the trachea; his present state of health not justifying the adoption of further measures for his relief.

This case is related to show the rapidity with which, under

some circumstances, granulations will collect around the tracheotomy tube, obstructing the passage of air through the larynx. I cannot but think, also, that it is worthy of remark that the ability to breathe through the larynx, which the child possessed in a limited degree, and with occasional interruptions, up to the time of the operation, was at once and entirely lost by the introduction of the tracheotomy tube; for from the date of the operation he was unable to inspire at all through the natural channel; though, as usually happens, the expiratory power was not altogether lost. This phenomenon may frequently he observed in those who have had tracheotomy performed for laryngeal obstruction, namely, that what little power of inspiration through the larynx the patient possessed before the operation, is at once, for a time at least, lost, by the introduction of the tube into the trachea.

Case 2.—Fanny S—, æt. 4, was admitted into the Children's Hospital, November 15th, 1863, under Dr. West's care. She was suffering from diphtheria, having the characteristic exudation about the fauces, with albuminuria. Tracheotomy was performed four days after her admission, by Mr. Marsh, the house surgeon, the child at the time being apparently moribund. The operation was thoroughly successful; four days after its performance the tracheotomy tube was removed; the canula having been previously closed for some hours to ascertain that the larynx was permeable. Within a minute or two of the withdrawal of the tube the rima glottidis seemed suddenly to close, and the child ceased to breathe until the canula was replaced.

From time to time, during some weeks, the same experiment was tried, and always with the same result. In the spring of the following year the child went to a convalescent institution, still wearing the canula.

After her return to London, in April, 1864, Dr. West asked me to see her.

It was now more than four months since the operation; and I could not discover, after repeated examinations, any com-

munication between the cavity of the larynx and the wound in the trachea. A solid mass of granulations seemed to have collected around the upper part of the tracheotomy tube, which completely prevented the passage of air. The child could not bear the canula closed for more than a few seconds, and if it were removed from the wound, the latter quickly began to contract, the edges inverting with each inspiration, so that in a minute or two it was difficult to replace the canula.

Under chloroform, which was administered through the wound, I succeeded in forcing a probe from below through the granulations into the cavity of the larynx; this was followed by a small elastic catheter, which was tied to the canula. Three days afterwards a very small tent of the sea-tangle weed was passed from below, and was followed in a few days' time by a larger tent of the same material. This plan was pursued until there was a free communication between the larynx and trachea of sufficient size for respiratory purposes.

The child could now bear the canula closed during the whole day, while during the night it was left open; under chloroform the finger could be introduced through the upper opening of the larynx so as to meet a director or bougie passed from below through the wound in the trachea; there appeared to be no obstruction whatever to the passage of air, and by this time also the child had so far recovered her voice as to be able to speak in a loud harsh tone when the canula was closed.

Under these circumstances the tube was frequently withdrawn from the trachea, but it always had to be replaced within a few minutes, on account of impending asphyxia. At the very first inspiration after each withdrawal, the edges of the wound began to invert, and in the course of three or four minutes the tube was replaced with difficulty. Thinking that perhaps the collapse of the wound in the soft parts over the trachea might narrow the calibre of that tube and thus account for the difficulty of breathing without the canula, a gutta-percha plug was made to fit the orifice of the wound and prevent its contraction when the canula was withdrawn.

On the only occasion upon which this plug was applied, symptoms of asphyxia came on instantly, the child being unable to make a single inspiration with it in the wound. For a month or two all treatment was abandoned as the child caught the measles.

September 20th, 1864.—Ten months after the operation the child was again examined; the opening between the trachea and larynx was found just large enough to admit a probe with difficulty; the voice was reduced to the smallest whisper, and the child could not bear the canula closed. By means of seaweed tents introduced through the wound, the communication between the trachea and larynx was again dilated to a fair size, so as to admit of the canula being kept constantly plugged during the day-time. As had occurred before, all attempts to keep the tube closed at night were ineffectual; so soon as the child fell asleep, if the canula were closed, a violent attack of dyspnœa would come on, and, if sufficiently conscious, she would herself withdraw the plug.

In November, eleven months after the operation, the patient was in much the same condition, except that under the application of powdered alum to the lower part of the larynx the voice had improved remarkably, both in power and tone. In order to free the mucous membrane as much as possible from irritation, the external canula tube was cut short, so as only just to enter the trachea, and this, with a cork in its orifice, was worn during the day, while at night-time the cork was replaced by an internal canula tube of the ordinary length.

By closing the orifice of the external tube with a piece of thin glass, so as to prevent the escape of air through it, one could examine with the ophthalmoscope the fissure leading from the trachea to the larynx. From the shape of this opening and the position assumed by its borders during speaking and inspiration, I felt convinced it was the under aspect of the rima glottidis, and that the parts were quite free from granulations. There could now be no obstacle to the passage of air through the larynx, except such as might be offered by the muscles of the glottis. I therefore came to the conclusion

that the *difficulty* of breathing through the larynx arose from a paralysis, or some imperfection of action either in time or power, of the dilator muscles, the crico-arytenoidei postici, and that the *sudden* symptoms of asphyxia which were wont to come on during sleep were due to a spasm of those muscles which close the glottis; *i. e.*, arytenoid and crico-arytenoidei laterales. In fact, the whole muscular apparatus of the larynx seemed thrown out of working gear.

It is worthy of remark that though the child could breathe freely with the canula closed, yet she could not bear its entire removal for more than a minute or two without suffering greatly from dyspnœa which soon became insupportable. And again, that the tube could be kept closed at all times except during sleep.

I venture to suggest the following explanation of these circumstances—that the mental agitation caused by the dread of impending suffocation which the removal of the canula produced, was sufficient to derange the normal action of the laryngeal muscles, so as to prevent the regular dilation of the rima glottidis at each inspiratory act.

It might be also that the influence of the will was necessary to regulate and secure the due action of these muscles, the perfection of whose movements had been impaired; and that on this account inspiration through the larynx during sleep was impossible.

The threefold office of the larynx—as the organ of the voice, as a conduit for air, and as the portal of the respiratory cavity—necessitates in its intrinsic muscles various and complex functions. They have to act both as vocalizers and as the janitors of the passage to the lungs, in the latter capacity either maintaining the patency of the canal, or more exceptionally excluding foreign bodies or noxious vapours by closing the rima-glottidis.

It would seem that the operation of tracheotomy in a manner mars the perfection of the coincidence between the action of the laryngeal muscles and those of inspiration; that it is liable to cause undue irritability and disorderly action of the muscles of the glottis, and to interrupt their

usual rhythm. Perhaps this is the effect of the enforced cessation of all exercise of function in the larynx which the operation entails; or, less likely, it is a reflex effect of the irritation caused by the canula.

These views are, I think, supported by the sequel of the case above quoted, as well as by others to which reference will be made hereafter.

When thirteen months had elapsed after the performance of the operation on Fanny S—, Dr. West kindly drew my attention to the account of a similar case by Dr. Sendler,[1] of which the following is an abstract:

"A child aged one year and nine months had tracheotomy performed on the sixth day of membranous croup; the operation gave immediate and permanent relief, but was followed by an inability of the larynx to resume its functions. The first attempt to remove the tube was made on the fifth day after the operation: the larynx being free from any permanent obstruction, the attempt was unsuccessful as were several subsequent ones. Before long, the child could bear the canula plugged at all times, except during sleep, but still it was impossible entirely to remove it, as on each occasion dyspnœa came on, increasing in severity until the tube was replaced. In the last attempt the child nearly lost its life, thereupon Dr. Sendler consulted Trousseau on the case, who replied as follows:

"'I have had two infants who have retained the canula, the one for three months, the other for seven months; in both cases I believed that a chronic inflammation of the larynx had followed the croup, and twice also, as it has been pointed out by M. Passavant, I was only able to remove the canula on the forty-second and fifty-third day of the disease; which latter seemed to remain in a subacute form for a long time.

"'At the time I observed these facts, my attention had not as yet been drawn to the partial or general paralysis which follows diphtheria, and now I am tempted to attribute (as you do) the extreme difficulty of inspiration to the para-

[1] 'Prager Vierteljahr.,' vol. xxxiv, p. 57.

lysis of the posterior arytenoid muscles. Nevertheless, when this paralysis exists, it causes a difficulty in deglutition, which you do not mention—this difficulty causes the entrance of fluids into the larynx during swallowing, and here is one of the most formidable accidents which follow tracheotomy.

"'Now if your patient drinks fluids easily, one must believe in the existence of a chronic inflammation of the aryteno-epiglottidean folds. Whatever it be, I am persuaded that the child will recover, and I advise you, whenever you can, to leave a closed canula in the trachea until the child has breathed for ten days in a natural manner. The canula with a dorsal opening is a useless and often dangerous instrument. For if you compare the calibre of the canula with that of the trachea, you will see that the section of the trachea is more than double the area of that of the canula; the air, therefore, can pass freely between them. On the other hand, the tissues project through the dorsal opening of the canula, and thereby the inflammation is kept up. Be therefore hopeful, and be sure that unless there be necrosis of the cartilages of the larynx (which is but little probable), the child will recover.'"

After the receipt of Dr. Trousseau's reply, Dr. Sendler allowed the canula to remain three weeks without any attempts to remove it until the child could bear it closed throughout the day, and for an hour or two at night-time. The canula was now again entirely withdrawn; but during sleep, the first night after its removal, such urgent dyspnœa came on that it was replaced. After the lapse of a month three unsuccessful attempts were made at short intervals to withdraw the canula. During the next six weeks no attempt was made to remove it, but its orifice was kept almost constantly closed, for a short time even during sleep, and the size of the instrument was gradually diminished until it was finally removed on March 11th, four months after the performance of tracheotomy. On this occasion Dr. Sendler determined to reintroduce the tube only under the most pressing emergency.

Throughout the day on which the canula was removed, no

difficulty in breathing was experienced, but at night-time soon after the child fell asleep, dyspnœa came on, increasing in severity until 11 p.m., when it attained its maximum of intensity, and the child's life seems to have been in some danger. From this time, however, an occasional pause in the severity of the symptoms took place, and by the morning all cause for immediate anxiety had passed away, and the child slept quietly. On the second day after the removal of the canula the wound had closed, and the child suffered no further inconvenience.

Acting on Dr. Sendler's experience in this case, on January 13th, 1865, *fourteen months* after the operation, I removed the tube from Fanny S—'s trachea, and covered the wound with a piece of plaister. This was done in the morning, about 9.30 a.m., and the child was placed for the whole day under constant observation, lest sudden need should arise for the introduction of the canula. Great care was taken to prevent any hurried breathing, and she was not allowed to play with the other children, or even to speak, as the increased demand on her respiratory powers seemed to threaten a laryngeal spasm.

She passed a tranquil day, the wound by evening had contracted to less than half its ordinary dimensions. During sleep that night she had a slight attack of dyspnœa, which soon passed off; next day the wound had nearly closed, and she could speak very fairly, though the larynx seemed not sufficiently free to allow of continuous conversation. The passage for air seemed also too small to allow of any muscular exercise, as the necessity for more rapid breathing caused the child distress. After running upstairs, the respiration was hurried, noisy, and difficult; but her respiratory power seemed quite sufficient so long as she was free from any unusual excitement; she could speak well. By the 17th of January the wound was quite closed, and the breathing was not distressed; on the evening of this day the eruption of varicella began to appear on her body, and towards night difficulty of breathing came on; it increased in urgency throughout the night, until Mr. Marsh, the house-surgeon (whom I had

advised to avoid the reintroduction of the tube if possible), found it necessary to give chloroform, having in view the strong probability that the obstruction to breathing was partly of a spasmodic nature.

The anæsthetic relieved the dyspnœa in a degree, but after a while the latter became more urgent, and at about 4 a.m. on the 18th, six leeches were applied over the larynx; the bites bled freely and gave much relief. Towards morning, the dyspnœa gradually subsided, and when I saw her at 11 a.m. it had in a great measure passed away; though the child was prostrate from loss of blood, anxiety, and fatigue. On this occasion I believe that the preservation of the child's life without the performance of tracheotomy, was entirely due to Mr. Marsh's active and judicious treatment.

January 18th.—Towards evening on this day, the dyspnœa again returned, and by midnight the child was in extreme danger from suffocation. The house-surgeon remained with her during the whole night, ready to reopen the trachea at a moment's notice. Towards the morning of the 19th the difficulty in breathing began to subside, and during the whole of that day a gradual improvement took place in the general as well as in the local symptoms. The night of the 20th she passed through with but little dyspnœa, and on January 21st, eight days after the removal of the tube, her breathing was quite easy, and her voice better than it had ever been before. A few days after this she was discharged well.

The following case furnishes another striking example of a permanent impairment of function in the laryngeal muscles after tracheotomy.

I am indebted to Dr. Russell Reynolds for permission to publish this case, and to Dr. S. Ringer for the notes of the same.

Case 3.—Arthur H— was admitted into University College Hospital under Dr. Reynolds, December 13th, 1863. The child was suffering from diphtheria, and the same evening tracheotomy was performed by Dr. Ringer; this gave speedy relief to the symptoms of asphyxia, and was soon followed

by a general improvement in the child's constitutional condition. The case subsequently progressed favorably, and on December 27th, a fortnight after tracheotomy, the tube was removed; it was, however, at once replaced, as without it the child could not breathe. After this it was frequently removed, and sometimes (especially latterly), it could be kept out for an hour or two at a time; but for longer than this it could not be dispensed with.

From time to time the child suffered from severe and even dangerous attacks of dyspnœa from the blocking up of the tube. Under a recurrence of these paroxysms his health began to fail, and in April of the following year it became evident that the dorsal part of the spine was carious, there being extreme angular curvature. Shortly after this the child died of bronchitis.

On post-mortem examination, two small ulcers were found on the mucous membrane of the trachea, just above the incision; these very slightly narrowed the calibre of the air-tube; the vocal cords and cavity of the larynx were found perfectly healthy. "The slight contraction of the trachea," says Dr. Ringer, "was insufficient to cause any difficulty in breathing." No paralysis of any part of the body could be detected at any period of the child's illness.

I venture to include this case in the same category as that of Fanny S—, attributing the inability to breathe through the larynx to a loss or perversion of function in the muscles of the glottis, and the result of the post-mortem examination quite confirms this view of the case.

Case 4.[1]—Dr. George Buchanan, of Glasgow, on March 23rd, 1864, performed tracheotomy on a child, aged three years, who had been suffering for some days with symptoms of diphtheria. The operation was perfectly successful, and the child made so rapid a recovery that on March 27th, four days after the operation, the tube was removed, and he was left breathing quietly and freely, partly through the larynx

[1] 'Brit. Med. Journal,' Sept. 7th, 1864.

and partly through the wound. Next morning he was quite well, being lively and playful, and taking his food with an appetite. At midday, whilst running about the room, he suddenly felt a choking sensation, became very pale, and died in a very short time, just after his medical attendant arrived. The wound seemed free from obstruction; the exudation had disappeared from the fauces altogether—the cause of death was not ascertained.

It seems highly probable that death in this case occurred from a spasmodic closure of the rima glottidis. The result suggests the propriety of a tentative and cautious removal of the canula before its final withdrawal, lest, after the subsidence of the original cause of obstruction, there remain a dangerous irritability of the laryngeal muscles.

Chassaignac in his 'Leçons sur la Trachéotomie,' p. 84, recommends that after the operation the canula should never be removed until three days have elapsed, and that when it is desirable to withdraw the canula, it should be done by degrees, and should be kept out of the wound for gradually increasing intervals of time.

In Cases 2 and 3 I have attributed the inability to inspire through the larynx to some imperfection in the action of its muscles, for the following reasons:

The larynx in each case was proved to be pervious to air by the ability to speak on the temporary closure of the orifice of the canula, and the patients acquired the power of breathing through the larynx for long intervals of time; in fact, at all times except during sleep, provided that the canula was in the wound. On the removal of the canula, the power of breathing through the larynx, though continuing for a time, was suddenly and almost completely lost; while in one case the larynx was ascertained to be free from any permanent obstruction by post-mortem investigation; in another by tactile and visual examination. The imperfections of action in the laryngeal muscles, to which allusion is made, include losses of rhythm, losses of power, or an undue excitability and proneness to spasmodic contraction.

As has been before remarked, it is possible that the ope-

ration of tracheotomy could produce such results in two ways, either by the sudden cessation of all movements in these muscles which it enforces, thereby interrupting the regular rhythmal action of the dilators of the glottis; or by a reflex irritation or paralysis of the same muscles from the continued contact of the canula with the mucous membrane of the trachea.

The paralysis of the muscles of the extremities as a sequel of diphtheria is not of infrequent occurrence. Muscular anæsthesia and paralysis of the velum palati; disorderly action of the pharyngeal constrictors; loss of vocalizing power in the muscles of the larynx; any or all of these may follow diphtheria. It might not, therefore, seem unreasonable to attribute to the same cause the disordered muscular action which occurred in the cases related above.

But supposing this view to be the true one, it is remarkable that the laryngeal muscles should be the only muscles affected, and that none of the cases above related, nor indeed any that have occurred in the Children's Hospital have suffered from any other form or degree of diphtheritic paralysis within the memory of the present medical staff.

Again, as opposed to this view, is the fact that the same temporary loss of function in the muscles of the larynx is liable to occur when tracheotomy has been performed for acute laryngitis, and that immediately after the performance of tracheotomy there ensues in almost all cases, for a time at least, a complete inability to inspire through the larynx.

In the following case the operation was performed for laryngitis:

J. J—, æt. 24, was admitted into St. Bartholomew's under Dr. Jeaffreson's care, January 4th, 1865. He had been suffering with symptoms of laryngitis one week. On the day of his admission the house-surgeon, Mr. Pearless, opened the trachea, and gave complete relief to all the most urgent symptoms. Fourteen days afterwards the canula was coughed out, and was at once replaced.

I saw him February 7th, one month after the operation;

he had for the last two days been wearing occasionally an instrument for directing the expired air through the larynx; no attempt had been made to remove the canula, which latter he could not bear closed for more than a minute at a time. If its orifice were closed after drawing about fifteen or twenty inspirations through the larynx, with some difficulty and great anxiety, the glottis appeared to close suddenly and completely, and the air had at once to be readmitted through the wound. With the above-mentioned instrument in the canula his voice was fairly good in all but tone.

A laryngoscopic examination of the upper part of the larynx showed the mucous membrane to be highly congested and somewhat swollen. In the only view I could get of the vocal cords, the metallic lustre of the tracheotomy tube was so distractingly evident that I could not speak with any certainty of their condition. A few days after this he was attacked with fever, and was not again examined until March 27th, when convalescent.

March 27th, three months after the operation, he was exactly as when examined before his attack of fever. There was a fair power of voice, but no use of his larynx as an air-passage to the lungs. To free the mucous membrane from irritation, the long tube that he had worn since the operation was removed, and a much smaller and shorter one was introduced, so as only just to enter the trachea. Two days after this he bore the canula closed for five minutes, and on April the 3rd for nearly ten minutes; but on each occasion the attempt to keep in the plug was terminated by a sudden and complete closure of the glottis.

On April 3rd a view of the interior of the larynx was obtained; the aryteno-epiglottidean folds were slightly reddened and a little puffy. The vocal cords looked natural, and although from the difficulty of examining the patient and from the cloudy state of the sky, I obtained only one clear view of the rima glottidis, yet there seemed to be a free passage for air.

On examining the wound the communication between the larynx and trachea, though somewhat reduced in size, ap-

peared quite sufficient for the purposes of respiration. The result of this examination, together with the sudden and choking kind of dyspnœa which attacked the patient on each occasion the canula was closed, make me believe that the difficulty of inspiration is due to an exalted irritability of the muscles of the glottis, and not to any permanent obstruction of the canal.

The rapid and abundant growth of granulations about the tracheotomy tube in the cases of George and Fanny S— furnishes an additional reason, if any were needed, for early and repeated attempts to withdraw the canula so soon as the original cause of obstruction is at an end, though on this point so high an authority as M. Chassaigne remarks that "there may be inconvenience attached to a too early removal of the canula, and there can be no harm in leaving it in."[1] [2]

The following case, related by Dr. Steiner, furnishes an example of the entire obliteration of the canal of the larynx after tracheotomy:

Case.—W. A—, æt. 2, a rickety child, had tracheotomy performed for croup July, 1862. The child recovered from the symptoms of croup, but every attempt to withdraw the canula from the trachea was immediately followed by symptoms of impending suffocation, and this condition of things continued to the time of his death, nine months after the operation, of hydrocephalus. On examination it was found that the opening of the larynx had entirely disappeared, the canal being perfectly obliterated by a firm and almost cartilaginous cicatrix mass which, of course, could have been produced only by deep ulceration following on the croupal exudation.

From the account of this case, as published in the foregoing report,[3] one would almost feel in doubt whether ad-

[1] 'Leçons sur la Tracheotomie,' p. 84.

[2] The retention of the canula in the wound may produce ulceration of the mucous membrane, or necrosis of the cricoid cartilage or tracheal rings.

[3] This case is published in the 'Prag. Vierteljahrschrift.' The account of the post-mortem was furnished me by Dr. West, who received it from Dr. Steiner.

hesion had taken place between the opposed surfaces of the vocal cords rendered bare by ulceration, as being the only spot in the larynx where adhesion could take place between its opposite walls; or whether, as nearly happened in the cases of Fanny and George S—, the canal of the larynx had become obliterated by a mass of organized granulations.

From the consideration of the foregoing cases it appears that among the obstacles to the re-establishment of natural respiration after tracheotomy are the following:

1st. A narrowing or complete obliteration of the passage of the larynx by the growth of granulations above and around the canula.

2nd. An impairment or complete loss of those functions of the muscles of the larynx which regulate the admission or exclusion of air through the rima glottidis.

3rd. Adhesion of the opposed surfaces of the vocal cords.

Having in view the avoidance of the contingencies above enumerated, I would draw attention to an instrument invented by Luer, of Paris, to be worn in the orifice of the canula as soon as practicable after the performance of tracheotomy.

This instrument, by means of a bullet-valve, ensures a free entrance of air by the wound through the canula, while it obliges all air leaving the cavity of the chest to pass out through the larynx.

The advantages which I think we may reasonably expect from such an instrument are the following:

1st. Its occasional and early employment favours the expulsion of false membrane and tenacious mucus from the cavity of the larynx during coughing.

2nd. Later in the case it may serve as an effectual and safe means of ascertaining the permeability of the larynx to the passage of air.

3rd. It restores the power of speech, and enables the laryngeal muscles to exercise at least some part of their ordinary functions, so as to prevent the injury they would suffer by complete disuse.

4th. By accustoming the larynx to the transit of air through

its cavity, it may diminish the irritability of the intrinsic muscles.

M. Luer's instrument (specimens of which I beg to place before the Society) quite fulfils the most important ends; while its costliness, weight, the want of simplicity in the manner of its attachment to the canula, and the fact that it requires a special canula for its adaptation, have interfered with its extensive employment.

I have therefore employed a more simple means for attaining the same end. This little appliance (specimens of which I beg to place before the Society) is light and inexpensive, and can be adjusted to any canula of corresponding size.

For the bullet valve of Luer is substituted an india-rubber flap, and its attachment to the canula is secured by a plug-shaped extremity. It is made by Messrs. Fergusson, of Giltspur Street, and it will be found to answer all the purposes of Luer's instrument.

THE APPLICATION OF SUTURES TO BONE

IN

RECENT GUN-SHOT FRACTURES,

WITH CASES;

ALSO REMARKS ON THEIR SIMILAR USE IN SOME OTHER FRACTURES AND OPERATIONS.

BY

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The dangers incident to the necessary transportation of gun-shot fractures from the battle-field are usually greater on long campaigns than those belonging simply to the wound *per se*.

Although from the beginning the advantage of primary operations seemed to be generally appreciated during the early part of the recent rebellion, there was, perhaps, a dangerous care to operate only in compliance with the immediate demands of the case; for in the official consolidated statement for gun-shot wounds for September, October,

November, and December, 1862, published by the United States Medical Department, we find that out of 228 cases of excision of the upper extremity, 195 are reported as secondary operations; while out of 521 amputations, 401 were secondary. There is every reason to suppose that the greater part of these secondary operations would never have been called for could the patients have remained at rest. Transportation, however, being inevitable, the successful surgeon learns to anticipate the innumerable contingencies growing out of it, and to adopt that present course which shall avert the greatest ultimate danger.

The tendency of the military surgeon to rush disheartened from extreme conservatism to reckless amputation has arisen mainly, not only from humiliating failure in saving the limbs, but in too many cases from the sad fear of having, in the most careful attempt to save their limbs, unfortunately sacrificed the lives of certain patients. The commonest cause of such failure has been the extreme difficulty, I may add the impossibility, of preventing such painful and dangerous motion of the fractured limb during transportation.

As it seemed hopeless to anticipate an improvement in the mode of transportation, the only attempt which seemed practicable was so to improve the condition of the limb as to diminish the motion of the fractured ends of the bone *en route* to general hospital.

Humanely anxious to preserve what often cannot be saved, the surgeon may direct a gun-shot fracture of the humerus to be put up firmly in splints, and the patient to be placed in an ambulance; but let the dressings have been applied ever so well, from the jarring and jolting, through rough fields and woods, they soon become disarranged; the previous firmness of the dressings but increases the now unequal pressure and constriction about the swelling limb, which usually cannot receive the necessary attention during the march.

The inflamed and strangulated wound, containing innumerable loose fragments and spiculæ of bone, which for successive days, perhaps, are being ground together between the

sharp-fractured ends of the shaft, is so much further irritated by the constant mangling of the soft parts that it is not uncommon to see a patient after a few days in the ambulance beyond any hope even from amputation.

Removal of the loose fragments and resection of the fractured ends of the shaft of the humerus has frequently been resorted to, but the chief difficulty still remained. The inevitable motion came, and the surgeon receiving the case at the general hospital would denounce the treatment as barbarous.

Hoping to steer equally clear of Scylla and Charybdis, I communicated to the Surgeon-General of the United States Army in 1863 an official document, in which I proposed a treatment specially for gun-shot fractures of the humerus, which, wherever applicable, would, I thought, supply the desideratum so generally felt.

It consisted in cutting down upon the seat of fracture, as in ordinary resection, removing all the fragments and spiculæ of bone, making a clear section of the fractured ends of the shaft, effecting perfect apposition, maintaining it by metallic sutures, and with the assistance of a light splint securing both *apposition and rest* during all the contingencies of transportation from the battle-field to general hospital.

The details of the operation are exhibited in the following case:

On the 12th of May, 1864, during the Battle of the Wilderness, Virginia, I was requested by Surgeon-Major Hubon to see Private Meisner, Company E, 52nd New York Volunteers. A Minié ball had perforated his left arm, producing oblique fracture of the middle of the shaft of the humerus, accompanied with severe though not extensive comminution. With the kind assistance of Surgeon Hubon, I enlarged the wound of entrance, making a straight longitudinal incision as external and dependent as possible, and as small as was consistent with the subsequent steps of the operation. Having removed all the detritus and loose fragments, the ends of the bone were turned outwards carefully enough to avoid peeling up of the periosteum, while with a metacarpal saw I made a section, removing the ends of the bone at such parallel ob-

liquities as to create the least possible shortening compatible with clean-fitting surfaces. After satisfying myself that, while the ends were in perfect apposition, there was no deflection of the line of axis of the bone, I drilled two pairs of holes obliquely through the ends of the bones as nearly as possible in the same line of diameter of the bone.

Through each pair of holes I then passed a suture of stout annealed iron wire, and secured them by twisting, as with silver wire in other parts. The ends were then nipped off short and turned down out of the way over the line of junction.

Fig. A.

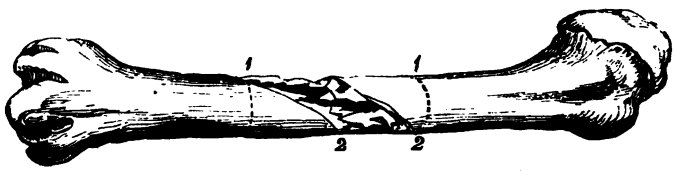


Fig. B.

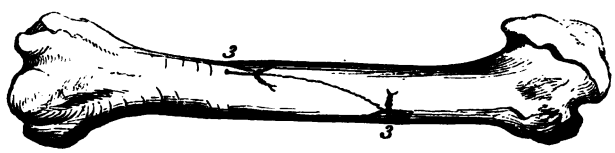


Fig. A.—1, 1. Shows length of fracture. 2, 2. Amount of bone removed.
Fig. B.—Condition after operation. 3, 3. Line of junction, with the two sutures used.

The amount of bone removed was considerable, from the obliquity of the section, but the shortening was not more than an inch, as seen in figs. A, B. The edges of the wound were, therefore, readily approximated with sutures.

A straight splint was then applied, extending from the axilla to the tips of the fingers, which was kept in position by a simple retentive bandage only, fully exposing the wound,

which the patient was directed to keep wet with water from his canteen.

From the 12th of May the patient was left in the forest in the hands of the enemy, without any attention or shelter, until the 17th, when he was transported about fifteen miles to Fredericksburgh. On his arrival the wound was full of maggots; these were cleared away and an examination made, when I was pleased to find that, notwithstanding these most trying circumstances, the ends of the bone were perfectly intact. From that time I attended the patient daily up to the 21st, during which time his pulse ranged from 88 to 96; he had a good appetite, and slept soundly. After the 19th there was no undue congestion of the wound, and the pus discharged continued moderate in quantity, and of a fine healthy character.

After the patient was sent north I was unable to obtain further particulars of his case, but I subsequently received an official statement from his commanding officer, to the effect that he had returned to duty and had since been transferred to another regiment.

As regards the manner of performing the operation.

The *incision* should be as small as is consistent with the subsequent steps of the operation, it being of primary importance that there should be the least possible disturbance of the soft parts; especially does this hold true with the periosteum.

The incision should be as dependent as possible, to allow of ready drainage of subsequent discharges.

If I have found a sound periosteal covering to any of the bone to be removed, I have dissected it up and afterwards brought it down over the line of junction.

The *drill* which I invented for the performance of this operation in the field, a description of which was published in the 'American Journal of Medical Sciences' for April, 1865, has the advantage over others in this operation in its being

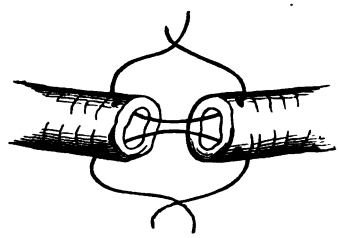
so small that it can be used with more facility without turning out so fully the ends of the bones. It works more rapidly, has no prominent points which can become entangled with the tissues, and the drills, fitting into the locket with a bayonet fastening, are in no danger of sticking fast to the bones and remaining behind on attempting to withdraw them. It also affords advantages for drilling at any desired angle with the surface of the bone. The drills are only two sizes larger than the sutures to be used.

It is important that the holes be drilled not through and through each end of the bone, but through one thickness only of the cortical portion each time, boring obliquely from without inwards twice through each end (*vide* illustration figs. D and C), so as to form two corresponding pairs of holes

Fig. C.

Appearance after application.

Fig. D.



Mode of applying the sutures.

as nearly as possible in the line of the diameter of the bone; the advantages are that only half as much bone is included within each loop of the suture, and may thus be extruded in half the time otherwise required; or, if desired, its removal may be much more readily affected. The two sutures, besides giving greater strength than one if they be introduced in the line of the diameter of the bone, they prevent any hinge-like motion and consequent danger of snapping.

The sutures employed were of annealed iron wire, the largest I could find having the necessary pliability. I think, however, that plated copper wire would be preferable, if it is intended to remove them after union, or should it be intended

to leave the ends protruding at the wound, so that the natural process of extrusion may be aided by frequently giving an additional tightening twist, so as to hasten the absorption of the part contained within the loop.

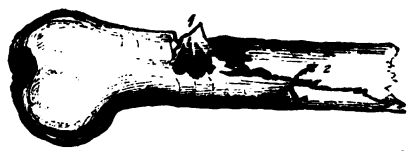
Contrary to any previous expectation, the circumstances of the following case led me to extend this plan of treatment to a case of fracture of the femur.

May 29th, 1864, I was called in consultation on the field to see Private I—, Co. B., 6th Ohio Cavalry. The patient, who was sixty years of age, and of broken-down constitution, had received a severe fracture of the femur from a large fragment of shell, which I found firmly wedged into the bone about three inches above the knee. From this point there proceeded a long oblique fracture, running upwards and outwards, with some comminution of the inner aspect of the bone in the vicinity of the piece of shell. The sharp-pointed lower fragment was overriding the upper, causing a shortening of about three inches, reduction of which, with such a wound could not possibly be maintained, with splints alone during transportation. Having made the requisite incision, and extracted the impacted piece of shell, I proceeded to remove the looser fragments; then, causing steady extension to be kept up while I kept my finger and thumb in the wound, I guided the fractured ends so that the corresponding serrations were accurately refitted to their original position for about two thirds of the circumference of the bone. Having somewhat enlarged the incision, I directed the limb to be so bent upon itself as to slightly reopen the fracture while I drilled the requisite holes, and passed a suture which firmly secured the interlocked fragments, rendering shortening or shifting impossible, as in fig. E. A straight splint was then applied, the wound being left exposed for the repetition of cold-water dressings. After the operation the patient ceased to complain of pain, and seemed very comfortable. The next morning he was hurried away in an ambulance train, and I saw no more of the case.

In consideration of his age, the nature of the wound, and his broken-down constitution, I was not surprised when sub-

sequently I incidentally heard that he died on the road three days afterwards.

Fig. E.



1. Impacted fragment of shell. 2. Suture retaining ends in apposition.

The shock of the operation involved in this plan of treatment, superadded to that of the original wound, immediately suggests itself, of course, as an objection to its adoption. Over against this, however, we must weigh the immediate dangers previously enumerated from the pressure and constriction of tight dressings attending the use of simple splints. Those from the enclosed fragments and spiculæ during transportation being in continual motion, and afterwards awaiting tedious extrusion by the slow processes of nature, keeping the patient in a condition which renders him continually susceptible to various intercurrent diseases, and, should union be ultimately effected, the degree of effort called forth for its accomplishment has left behind such a surplus of provisional callus, and other pathological changes in the parts adjacent, as seriously to diminish the usefulness of the limb for a long period, with a still further danger that after apparent recovery tertiary sequestræ, as mentioned by Dupuytren, may continue to be extruded for ten, fifteen, or even twenty years thereafter.

In the operation described the chief causes of the evils above enumerated are removed. *Its advantages consists mainly in this:*

1st. *Negatively.—In the absence of everything which may act as a foreign body and dangerous source of irritation.*

2nd. *Positively.—In securing complete coaptation and perfect rest.*

This plan of treatment is new only in its application. Sub-

stantially the same operation, including the use of the wire suture, has for a long time been practised, and still meets with general approval, as a *secondary* operation for ununited fracture.

Other things being equal, why should it be less advisable as a *primary* one, where, in the absence of all pathological changes, we have a healthy condition of all the parts, with sound bone in apposition and at rest.

These considerations led me to hope for a healthy process of uninterrupted repair, with speedy use of the limb after union.

The selection of the cases (which at most must be quite limited in number, either on the field or in civil practice), the amount of bone which may be with propriety removed, &c., must, of course, be a matter of careful judgment.

As it will probably take a long time to collect reliable histories of the cases of this kind which I sent from the field to different hospitals in the various states during the campaigns of 1864, it would be unwise for me to anticipate what I hope to be able to state in a full report hereafter.

While taking occasion to bring this plan of treatment before the notice of this Society, I would suggest the similar use of wire sutures as a retentive appliance in certain cases in civil practice, especially in cases of compound fracture with great displacement, with or without section of the ends of the bone, where but little additional enlargement of the wound is necessary for the operation.

In Pirogoff's operation, should it be practised, where one suture would counteract the troublesome traction of the tendo Achillis.

In fracture of the jaw, where I have found it to answer admirably.

With or without section, as the case may demand, I consider that the use of sutures in the treatment of fractures is a valuable accessory of which the surgeon may sometimes avail himself with great advantage.

ON THE

PATHOLOGY OF TETANUS,

ILLUSTRATED BY CASES AND DRAWINGS.

BY

J. LOCKHART CLARKE, F.R.S., &c.

COMMUNICATED BY

DR. RADCLIFFE.

Received April 28th.—Read June 27th, 1865.

NOTWITHSTANDING the number of cases of tetanus in which the spinal cords have been dissected and carefully examined in the ordinary way, by different persons in different countries, no observer, I believe, has hitherto discovered anything like definite lesions, or anything, indeed, abnormal, except a general increase of vascularity or a variable and local congestion. Up to the present time, therefore, the *pathology* of this frightful malady has been involved in the greatest obscurity, and various are the speculations in which theorists have indulged, in the absence of positive knowledge, to explain its mysterious nature. In this communication, however, I shall show, beyond the possibility of doubt, that in six cases in which the cords were examined by myself, lesions of structure of different kinds, and frequently of surprising extent, were discovered.

The first case which I shall bring before the attention of this Society occurred in the North Staffordshire Infirmary. Its history, which was furnished me by Mr. W. Dunnett Spanton, is as follows:

"Joseph G—, æt. 46, single, a labourer, was admitted into the North Staffordshire Infirmary, October 22nd, 1864, for extensive burns.

"The patient, a strong, muscular, healthy man, had, while intoxicated, been sleeping near a lime kiln, and his clothes caught fire, causing deep burns of the face, arms, neck and chest. He was brought to the infirmary about six hours after the accident.

"On admission the burnt surface was dressed with a strong solution of nitrate of silver and cotton wool; he was ordered to take an effervescing mixture with ten drops of tincture of belladonna every four hours, and to have milk, beef tea, and four ounces of wine.

"On October 28th delirium came on, the patient being very weak; the burns sloughing. Some carbonate of ammonia was ordered, and the belladonna omitted. An opiate was given at night.

"The man became weaker, the delirium continuing more or less up to the early morning of November 1st (ten days after admission), when trismus became marked. The patient complained first of some pain and aching about the muscles of the jaws and neck, and then experienced some difficulty in opening his mouth, which, in the course of about two hours, was so much increased that a spoon could not be put between the teeth. When fluid was poured between his teeth the patient had some difficulty in swallowing it. The delirium completely left him, and he appeared quite intelligent, replying to questions as well as his altered voice would permit.

At 10 a.m. of the same day, when an attempt was made to sit up in bed, opisthotonos came on, which quite prevented his doing so. Trismus was almost complete; there was no spasm of the extremities. Risus sardonicus was present in

some degree; skin was hot and dry; pupils equal, of moderate size, acting freely to light; pulse 130, full, soft; respiration 25, spasmodic, short; bowels acted the day previously; micturition free.

"Was ordered to take two minims of Fleming's tincture of aconite in a very small quantity of water every hour, the dose to be rapidly increased, and to have enemata of milk and brandy every four hours.

"2 p.m.—Risus sardonicus more marked; intelligence clear; trismus complete; the patient says that since he took the medicine the pain about the jaws has almost gone; opisthotonos when any attempt is made to move, but not constant; able to swallow a few drops at a time when poured between the teeth; no spasm of the extremities; pulse 130, soft, smaller; respiration 26, short, and irregular; passes water with difficulty.

"During the evening spasm of the limbs came on, with marked opisthotonos, the patient evidently becoming weaker. The dose of tincture of aconite was increased up to twelve drops, but was not taken so regularly as at first. The patient died at midnight, apparently from exhaustion, being quiet for a short time before death.

"A post-mortem examination was made twelve hours after death.

"Body warm; rigor mortis slight; over the face, neck, both arms, the anterior and right side of the chest, were the marks of a deep burn, the surface suppurating. Body well nourished; subcutaneous fat in moderate amount.

"The cellular tissue and muscles of the back, especially in the upper dorsal region, were deeply congested; there was no ecchymosis. On opening the spinal canal the membranes of the cord at the upper cervical region presented a redder appearance than elsewhere, and the vessels were more congested. The cut surface of the spinal cord, at the junction of the cord with the medulla, was of a pink colour. The cord was removed entire, and sent to Mr. Lockhart Clarke for examination.

"The brain was quite healthy in appearance, and firm.

There was a small quantity of clean serum in each lateral ventricle. The membranes were of a natural colour.

"The chest and abdomen were not examined."

Morbid anatomy.—The parts which were sent by Mr. Dunnett Spanton for my examination were preserved in a weak solution of chromic acid.

The medulla oblongata and the upper end of the cord which gives origin to the first cervical nerves were apparently free from disease. At the second cervical nerves, however, streaks and irregular areas of disintegration were observed in different parts of the gray substance, and particularly around the central canal, on the right side of which was a space of considerable size, containing a finely granular fluid, with the débris of blood-vessels and nerves. The posterior and lateral white columns, especially along the edges of the various fissures which transmit blood-vessels, were damaged in a similar way; and in some sections the deeper portions of the posterior columns which rest on the transverse commissure were softened to a considerable degree.

On descending through the cervical enlargement the lesions of structure in the gray substance continued to increase, and occurred chiefly behind and at the sides of the canal. In fig. 1, Plate IV, the posterior commissure between the bases of the horns was destroyed by a fluid, transparent and granular area (*a*), containing the débris of the tissue which it replaced, and bounded by the jagged or irregular edges of the adjacent tissue, which projected into it to different distances, and were readily observed to be themselves in process of disintegration. On the left of this transparent space, at the base of the posterior horn, the solution was only partial, for the softened substance was either infiltrated uniformly by fluid or eroded, as it were, irregularly by small but numerous points of fluidity. Close on the right and in front of the canal, amongst the decussating fibres of the anterior commissure, was another fluid space of considerable size, transparent, indeed, but granular from the débris of the fibres which it replaced.

In fig. 2 the posterior commissure behind the canal

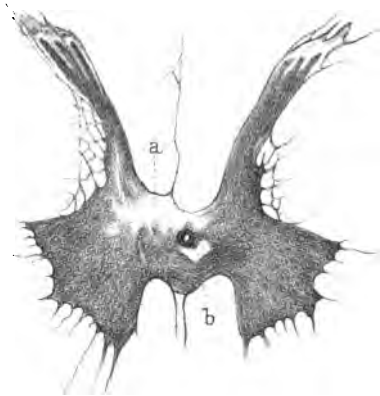


Fig. 1.

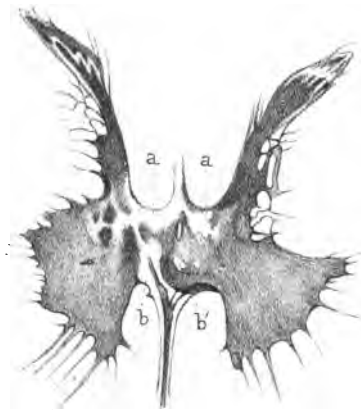


Fig. 2.

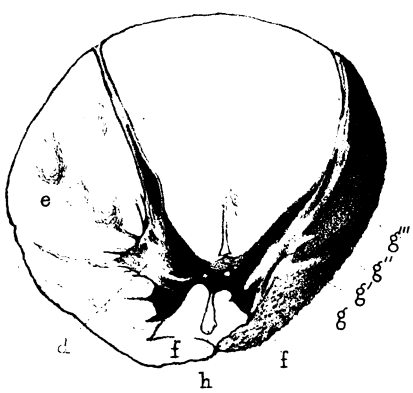


Fig. 3.

Fig. 4.

J. L. Clarke del. G. H. Ford lith. W. West imp.

was completely destroyed by two semifluid and irregular areas (*a*, *a*), which coalesced at different points, and on the left side one of these was continuous with a large and perfectly fluid space (*b*), which extended from behind forwards, quite across the gray substance, dividing the anterior commissure, and containing the remains of an eroded blood-vessel (*c*) which entered through the anterior fissure. On the right side the anterior commissure was further damaged by two separate but fluid spaces (*b*) of smaller size.

Throughout the rest of the cervical enlargement lesions were discovered like those I have just described, varying from a state of softening to a state of complete solution, and diminishing at intervals, or almost disappearing, to return shortly in the same form.

At the upper part of the dorsal region the shape of the cord was much altered, and extensive lesions were everywhere seen. On the left side, as shown in fig. 3, the lateral half of the gray substance (*d*) was softened almost to fluidity, and rendered exceedingly transparent. In the lateral column (*e*) were several elongated and crescentic areas of partial disintegration, and the superficial half (*f*) of the corresponding anterior column was softened to a considerable degree. On the opposite side the lateral portions of both the anterior and posterior gray substance were destroyed by long tracks or streaks of fluid disintegration (*g g′ g″ g‴*), and the remains of the posterior horn (at *g‴*) had fallen backwards and outwards with the antero-lateral column. This column was much softened, especially anteriorly, and in its deeper layers, which were reduced to a semifluid state and fused with the lateral part of the gray substance (at *g g′ g″*), so that the line of demarcation between the two kinds of structure—as seen on the opposite side—was wholly obliterated. The superficial portion of the anterior column (*f′*) had a kind of spongy or quaggy consistence, in consequence of the minute areas of fluid or semifluid disintegration which existed between its bundles. At the anterior median fissure the edges of the anterior column had been destroyed by the same morbid process, which resulted in the formation of a perfectly fluid

but finely granular space of a pyriform shape. At the posterior median fissure a somewhat similar appearance was observed, and in the posterior columns there were numerous irregular areas of partial disintegration, like those in the lateral column of the opposite side.

For the distance of a few lines the morbid appearances diminished both in extent and degree; but about half an inch lower there was some dilatation of the cord, in which the lesions were very similar to those represented in fig. 3; and just below this a considerable extravasation of blood was found in the posterior columns between the horns. At the fourth dorsal nerves the cord was again much swollen, chiefly along the posterior columns, and the gray substance was extensively damaged, as shown in fig. 4, in which the irregular blank spaces represent areas of fluid disintegration, more or less interrupted or traversed, in different ways, by masses and streaks of half-dissolved tissue. Moreover, an alteration in shape and a want of symmetry of the opposite sides were frequently observed. In fig. 4 the changes are seen chiefly on the left side, where the posterior horn is thrown outwards.

Immediately below this swelling the lesions of structure diminished considerably, although the gray substance contained many small spaces in different stages of disintegration; but after a few lines in descent the lesions again increased, and large extravasations of blood were found along the whole lateral part of the gray substance, on both sides in some sections, on one side only in others; soon, however, they entirely disappeared, and only a few small areas of disintegration remained; but after a very short interval they returned in the same form; and so, through the rest of the dorsal region, the different kinds of structural change alternating in extent and degree, sometimes existing together, sometimes singly, and at other times almost disappearing, were observed in different sections. At the lowest part of this region there was no hæmorrhage whatever, and the gray substance was less damaged than elsewhere.

It will not be necessary to describe in detail the morbid

appearances discovered in the lumbar region. It is sufficient to state that they bore a general resemblance to those found in other parts of the cord, particularly in the cervical enlargement.

The second case to which I shall call attention is one of idiopathic tetanus which occurred in St. Mary's Hospital. The notes of the history and treatment I have not yet received, but the spinal cord was sent to me for examination by Dr. Handfield Jones. For my present purpose, I need only say that areas of disintegration and exudation were discovered in different parts of the cord, but chiefly in the central gray substance.

The third case, which was of the traumatic kind, occurred in the North Staffordshire Infirmary. The spinal cord, as I received it from Mr. Dunnett Spanton, was much mutilated, and spoiled by long maceration in spirits of wine; but, notwithstanding, I was able to affirm with great confidence that in the lumbar region, at least, the posterior gray substance contained numerous streaks and irregular areas of disintegration. The case was published in the Lancet of June 3rd, 1865.

The fourth case, of which I have not yet received the history, died in the West Norfolk and Lynn Hospital. The spinal cord, which was sent to me by Dr. Lowe, I have only partly examined, but I have seen enough to convince me that it contains lesions of the same nature as those described in the first case.

The fifth and sixth cases occurred in St. George's Hospital. Only parts of the cords fell into my possession; but these presented the most unequivocal marks of structural lesions. Both cases were published in the 'Lancet' of September 3rd, 1864, with illustrations, and the preparations from which the drawings were made accompany this communication.

In these six cases I have given nothing but the actual pathological facts, without any theory or speculation. But these facts suggest certain questions which are very important in a physiological and pathological, but especially in a practical, point of view.

1. Are structural lesions present in those cases of tetanus

which recover? That they are *not* present, or are present only in a small degree or to a slight extent, is probable from the actual fact of the recovery, which certainly could not take place, at least without consequent paralysis, if the lesions were as extensive as those discovered in the above cases. It is not improbable that small exudations or even minute structural changes may have occurred, and been arrested in their course, and it would be interesting to ascertain whether, in cases of recovery, any paralytic affections or abnormal phenomena of motion or sensation remain after the attack.

2. Are the structural lesions or disintegrations of tissue the *effects* of the functional excitement of the cord manifested in the tetanic spasms? That they are *not* the effects of this excitement would appear from the following facts:—That they more frequently occur in the central parts of the gray substance around the canal, where the nerve-cells are scanty; that the nerve-cells of the anterior gray substance which give origin to the motor nerve-roots remain apparently unimpaired; that the structural changes commence frequently in company with exudations around or in the vicinity of blood-vessels, which are themselves commonly found dilated, and frequently in a state of disintegration; and, lastly, that they are exactly similar in kind to the lesions or disintegrations which I find in various cases of ordinary paralysis, in which there is little or no spasmodic movement.[1] It appears, therefore, that they result from a *morbid* state of the blood-vessels, and not from excessive functional activityof the cord.

3. Are the structural lesions of the nerve-tissue the *cause* of the tetanic spasms? That they are not the direct or sole cause may be inferred from the fact that in those cases of paralysis in which similar lesions exist they do not give rise to tetanic spasms or convulsions, although twitching of individual muscles or groups of muscles is not uncommon.

4. On what, then, do the tetanic spasms really depend?

[1] See 'Beale's Archives of Medicine,' No. IX, 1861; No. XIII, vol. iv. 'British and Foreign Med.-Chirurg. Review,' Oct., 1863, and especially April, 1864, and July, 1864.

Since the lesions of the cord in cases of paralysis in which there is commonly no spasm, are similar to those of tetanus, it follows that the latter disease, in regard to its morbid anatomy, differs from the former only in being associated with a morbid condition or injury of some of the peripheral nerves. It would therefore appear that this condition or injury of the peripheral nerves is the determining cause of the phenomena, and that the spasms of tetanus depend on the conjoint operation of two separate causes. First, that they depend on an abnormally excitable state of the gray nerve-tissue of the cord, induced by the hyperæmic and morbid state of its blood-vessels, with the exudations and disintegrations resulting therefrom. This state of the cord may be either an extension of a similar state along the injured nerves from the periphery, or may result from reflex action on its blood-vessels excited by those nerves. Secondly, that the spasms depend on the persistent irritation of the peripheral nerves, by which the exalted excitability of the cord is aroused; and thus the cause which at first induced in the cord its morbid susceptibility to reflex action is the same which is subsequently the source of that irritation by which the reflex action is excited.[1]

This explanation derives some confirmation from certain cases of paralysis. In one of these, which I published in the 'British and Foreign Medico-Chirurgical Review,' July, 1864, there was partial paralysis of one arm, and the lesions and disintegrations which I found in the cervical enlargement of the cord bore a very striking resemblance to those which I have described in the first case of tetanus recorded in this paper. So long as the arm was enveloped and kept warm in flannel it remained perfectly still, but whenever it was ex-

[1] In so-called idiopathic tetanus, arising from exposure to cold and damp, it is probable that the morbid condition of the blood-vessels of the cord results from changes in the state of the peripheral nerves, which may act through reflex action or otherwise. There are many facts recorded which tend to show that congestion and inflammation of the nervous centres are produced in a similar way.

posed to the impression of cold air it began to move in a spasmodic and jerking manner.

Appendix.

In the walls of the blood-vessels there was no morbid deposit nor any appreciable alteration of structure, except where they shared in the disintegration of the part to which they belonged; but the arteries were frequently dilated at short intervals, and in many places were seen to be surrounded, sometimes to a depth equal to double their diameter, by granular and other exudations, beyond and amongst which the nerve-tissue, to a greater or less extent, had suffered disintegration. We have reason, therefore, to infer that the lesions of structure had their origin in a morbid condition of the blood-vessels, resulting in exudations with impairment of the nutritive process.

Of the structural changes which constitute what I have called "granular" and "fluid" disintegration—whether they commence immediately around the blood-vessels or not—the first stage consists of a softening of the nerve-tissue. When a small portion of this tissue is very carefully placed on a glass slide, without further disturbance—even without covering-glass, to avoid compression—and examined under a sufficiently high magnifying power, a large number of the nerve-fibres are found to be more or less altered in structure. Their medullary sheaths, or white substance, are either ragged, puckered, wrinkled or granular; or they are partially stripped from their axis-cylinders and broken into fragments of different sizes, or formed into globular masses.[1]

In the second stage the nerve-substance becomes softer or semifluid, and more transparent. A greater number of the fibres are involved in the destructive process, and the frag-

[1] See also 'British and Foreign Medico-Chirurgical Review,' April, 1864, p. 491, and fig. 5 B.

ments of those that were first destroyed are reduced to finer particles and mingle with the granules of the exuded fluid. To this morbid condition I have given the name of "granular disintegration."

By a continuation of the same process the substance becomes reduced to a perfectly fluid state. Sometimes a fluid area of considerable size originates at a single spot, which gradually increases by solution of the surrounding tissue. This is more common around the larger blood-vessels. Sometimes it begins at several isolated spots, which advance towards each other and coalesce, in various ways, between irregular masses of the tissue, which either remain connected, more or less, with the surrounding substance, or are separated and left as islets in the fluid.[1] In such cases the gradual transition from granular disintegration to a state of solution may be very readily observed along the edges of the several masses. The fluid itself, however, as might be expected, is at first more or less granular, holding in suspension the fragments and particles of the disintegrated substance; but in many places it is perfectly pellucid.

The transition from the one condition to the other may be very strikingly observed in some isolated spots where the process of solution is most rapid from without inwards. In these cases, although the fluid at the circumference may be perfectly clear, it becomes not only granular, but more coarsely granular, on approaching the centre, where the process of solution is later. These isolated spaces are very often sharply circumscribed, as if the tissue had been suddenly dissolved by a drop of some solvent fluid. They are frequently seen around or at the sides of the blood-vessels, and are commonly more or less fusiform, oval, or circular.[2]

[1] See also 'British and For. Medico-Chirurgical Review,' July, 1864.

[2] See Beale's 'Archives of Medicine,' 1861, No. IX, pl. i, fig. 3; and 1863, No. XIII, pl. ii, fig. 1.

ON

MORBID CHANGES IN THE STOMACH

AND

INTESTINAL VILLI PRESENT IN PERSONS WHO HAVE DIED OF CANCER.

BY

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THE difficulty of accounting for the origin of cancer and the want of success in all our efforts to arrest its progress or effect its cure, render investigations upon it of general interest. Of late years inquiries have been chiefly directed to obtaining clearer views as regards the development and anatomical structure of the tumour itself. As far as I am aware, no attempts have been made to ascertain whether the progress of the disease is accompanied by changes in other important parts; and yet an accurate knowledge of the condition of the various organs connected with the formation of the blood must be necessary to enable us to determine whether a malady having chiefly a local manifestation originates in an error of local or of general nutrition. Until, therefore, we have carefully scrutinised all the glandular structures, we

cannot be certain that the predisposing causes of the so-called malignant action may not lurk in imperfect or perverted actions of some of these organs.

The microscope has shown us no local changes which *precede* the formation of cancer; consequently we are unable to foresee and powerless to prevent its invasion. If we could discover that anatomical or functional alterations take place in tissues other than those obviously affected, a careful observation of their conditions might warn us of the approach of the disease and we might be enabled to avoid any circumstances likely to excite its development.

In many cases of cancer we can scarcely account for the death of the patient. There is no secondary formation in any important organ, and the failure in strength has been out of proportion to the amount of local mischief, so that we are forced to admit either that the blood has been infected, or that some fatal change of a non-cancerous nature has occurred in the viscera.

But if all the laborious investigations into the nature of cancerous tumours have left us still ignorant of their causes, and of any structural changes preceding their formation, and if in many cases the cause of death is not apparent, is it not probable that the medical practitioner will derive more advantage from a knowledge of the changes which may be found to take place in the *vital organs* than from further inquiries directed exclusively to the locally diseased tissues?

I have, in the following paper, stated the results obtained from the microscopic examination of the stomach in fifty-seven, and of the intestines in twenty-three, cases of cancer.

In addition to the ordinary difficulties attending anatomical investigations, the liability of the stomach to peculiar post-mortem change has tended to produce differences of opinion amongst pathologists respecting the explanation of the morbid appearances that are to be found in this organ. Thus, Dr. Handfield Jones,[1] in an important paper in which he first drew attention to the pathological anatomy of the

[1] 'Medico-Chirurgical Transactions,' Vol. XXXVII.

stomach, describes *destruction of the secreting tubes* as of frequent occurrence. He attributes it to the pressure of "nuclear masses" (which he believes to originate in degenerative changes) situated below and between the tubes.

Dr. Fox,[1] on the other hand, in an interesting communication to this Society, whilst admitting the frequency with which the stomach is affected in various diseases, attributes the morbid changes *entirely* to inflammation, and states that he only met with one case in which nuclei were present below the tubes. I have constantly found the "nuclear masses" mentioned by Dr. Jones,[2] and I feel quite sure they were not the result either of accident or decomposition, since in every case the mucous membrane was previously hardened either by spirits or chromic acid, and in many instances the post-mortem examinations were made in the coldest months of the year, and in an unusually short time after death. I have not, however, found the gastric tubes destroyed in the splenic or middle regions of the stomach in non-cancerous diseases so frequently as Dr. Jones; but I have usually observed, in addition to the "nuclear masses," the appearances of chronic catarrh as described by Dr. Fox.

I have to regret that in the following cases I am unable to give the history of each patient and a description of the peculiarities of the local disease, or to report the manner in which digestion was performed during life. My opportunities make it necessary that I should limit myself to detailing the changes I have observed in the mucous membrane of the stomach and intestines.

Cancer of the Breast.

Observation I.—A woman, æt. 62, died of cancer of the breast in the Middlesex Hospital,[3] February 7th, 1865. The mucous membrane of the stomach was everywhere thin, es-

[1] 'Medico-Chirurgical Transactions,' Vol. XLI.

[2] In accordance with his views, I have described them as enlarged solitary glands.

[3] To avoid repetition, it may be stated that all the observations mentioned in this paper were made upon patients who died in the cancer wards of the Middlesex Hospital.

pecially along the smaller curvature; it was smooth, but much injected in patches. Along the greater curvature it was rather thicker, adherent firmly to the subjacent coat and mammilated. In this part the tubes were united together, and the spaces between and below them were loaded with nuclei and cells, nuclei also being everywhere visible in the muscular coat. The solitary glands were greatly enlarged, and mostly empty at the centre, but at their edges they presented thick layers of cells and nuclei. In the more wasted parts of the mucous membrane the remains of the tubes appeared as bulbs, or as irregularly shaped masses of fat and broken-up gastric cells, or as lines of fatty matter so small as to resemble blood-vessels, whilst their interspaces were occupied by fatty and granular matters. In some places no remains of the solitary glands could be perceived; in others elongated cavities surrounded by thin layers of nuclei and cells marked their former position. The muscular coat seemed to consist of little else than fibres. The duodenum was large, but to the naked eye showed no appearance of disease. Examined microscopically, Brunner's glands were seen to be enlarged and loaded with nuclei and fatty cells. The intestinal villi were normal.

Observation II.—A woman, æt. 37, died June, 1863. The mucous membrane of the stomach was very thin, and much congested in parts. At the cardiac end it was everywhere sprinkled over with small white spots about the size of a pin's head, that looked, but did not feel, as if raised above the surface. In this portion of the organ the membrane was converted into fibrous tissue, fat, and granular matters, whilst the tubes had disappeared or their remains only could be recognised as faint lines of fatty matter. The muscular tissue was thickened and fibrous, and the blood-vessels seemed very large and in many cases thickened and opaque. In a small portion of the organ the tubes could be distinguished, but they were firmly adherent to each other, and were filled with granular matter and a few large gastric cells.

OBSERVATION III.—A woman, æt. 43, died in July, 1863. In one part of the middle region of the stomach, the mucous membrane seemed hard and fibrous. The solitary glands were in some parts enlarged and the tubes distorted or withered, as in Observation i, but in other parts, although the solitary glands were not apparent, the tubes were firmly adherent to each other, their basement membrane was thickened, and the connective tissue between them and the muscular coat increased. The openings of the tubes on the free surface were larger than usual.

OBSERVATION IV.—A woman died August, 1863; no post-mortem digestion had taken place. In the cardiac region the tubes were greatly wasted, and for the most part their closed ends only remained. They were firmly united in every other part of the stomach, their interspaces being occupied by nuclei, as in Observation i. The gradual conversion of tubes into bundles of fibre was observable at many points.

OBSERVATION V.—A woman died August, 1863. In the smaller curvature of the stomach, no indication of tubes existed, excepting here and there a bulb surrounded by thick fibres; the whole of the structure being composed of cells, nuclei, and fibres. In the cardiac region the tubes were in a similar condition. The solitary glands were in many places very large, and loaded with cells and nuclei.

OBSERVATION VI.—A woman, æt. 63, died December, 1863. The stomach contained torulæ in large quantities. The mucous membrane was remarkably thin, but much congested; along the lesser curvature were patches of a white opaque appearance. The tubes could be easily recognised, but contained only small cells; between and below the tubes the spaces were loaded with nuclei.

OBSERVATION VII.—A woman, æt. 63, died December, 1863. The stomach was extremely thin everywhere, but

especially so in the cardiac region, which was not digested. A broad band of dense white tissue ran along the smaller curvature for two thirds of its length; excepting this the middle and pyloric regions were of a dark gray colour. The tubes could be distinguished in some parts of the cardiac region, but were united together, whilst in other places they were converted into bundles of fibres. In the white band no remains of tubes were visible, the whole tissue appearing to consist of fibres and granular and fatty matters. In the gray parts the tubes were firmly adherent, and intermixed with cells and nuclei. The solitary glands were much enlarged and filled with cells and nuclei.

Observation viii.—A woman, æt. 60, died January, 1864. The mucous membrane of the stomach was exceedingly thin and pale, but not digested. Towards the cardiac end the remains of the tubes appeared only as confused masses of fat and granular matter, and the membrane itself seemed opaque and granular. In other parts the tubes were firmly connected together, and their basement membrane thickened, whilst within and around them were cells and nuclei from $\frac{1}{3000}$ to $\frac{1}{4000}$ of an inch in diameter. Few normal gastric cells could be seen, but the solitary glands were greatly enlarged and filled with the same cells and nuclei.

Observation ix.—A woman, æt. 75, died July, 1865. The mucous membrane of the stomach was not atrophied; the tubes were adherent to each other, their interspaces being occupied by nuclei and cells. The tubes contained gastric cells, generally of a small size.

In all the above cases the appearances were similar. In all except one the mucous membrane was much wasted, chiefly at the smaller curvature and splenic regions; in one instance, in which I weighed the mucous membrane, it only amounted to 360 grains; it was usually congested in irregularly shaped patches, and the veins were generally of large size. When examined by the microscope they all pre-

sented evidences of disease, which were most perceptible where the membrane was thinnest. In the earliest stage of the disease the solitary glands were enlarged and filled with cells and nuclei; the gastric tubes and sometimes the muscular fibres were displaced by these bodies, which were also scattered everywhere through the membrane; the tubes adhered firmly to each other, but still contained normal gastric cells. Later, the solitary glands appeared empty in the centre, but surrounded by thick layers of nuclei; the tubes could no longer be traced in their whole extent, but could be recognised only as bulbs filled with fatty cells, or as lines of cells, whilst the whole tissue was obscured by fatty and granular matter. In the last stage the solitary glands had disappeared, and the tubes were replaced by fibres, the replacement of glandular structure by fibre being the ultimate metamorphosis.

These anatomical changes seemed to have produced a concomitant decrease of functional power. Post-mortem digestion seldom occurred even in the summer, and in one case, in which I digested ten grains of albumen for eleven hours in an acidulated infusion of the mucous membrane, only six tenths of a grain were dissolved.

In the next cases, although no nuclear deposits were present, the structure of the stomach was seriously injured by an increased formation of fibrous tissue.

Observation x.—A woman, æt. 56, died in August, 1864. The mucous membrane of the stomach was exceedingly thin. At the greater curvature was an irregularly shaped puckered patch of thin membrane, about three inches long by one in width, and from this projected fibrous bands in different directions, but not so well marked as is usual in a cicatrized ulcer: there was no change in the shape of the stomach. At the puckered part the membrane was pale, but on every side it was greatly congested. Where most contracted the mucous membrane seemed to be converted into a very thin layer of fibres, intermixed with a few cells; in other places the tubes appeared to be drawn in a

longitudinal direction, and were in process of conversion into fibres, the cells being absorbed and the thickened basement membrane constituting the fibrous bundles. Along the smaller curvature the tubes were easily distinguished, filled with gastric cells, but adherent to each other, their basement membrane being much thickened and the connective tissue below and between them greatly increased.

It might appear doubtful whether the appearances in the above case were not caused by the cicatrix of an ulcer, but the great extent of the contraction without alteration in the shape of the stomach itself seems to forbid the supposition. Degeneration was in progress in all parts of the organ, and in cases of simple ulcer of the mucous membrane I have not usually found much structural alteration at a distance from the sore.

Observation xi.—A woman, æt. 55, died January, 1865. The mucous membrane of the stomach was very thin, but not digested. In the middle and splenic regions the tubes could be distinguished, but were firmly adherent to each other; their basement membrane was much thickened, and they contained large fatty gastric cells; the solitary glands were not enlarged. The connective tissue below them was greatly increased, and the muscular coat seemed fibrous.

Although I did not detect any enlarged solitary glands in the two foregoing cases, nuclei were everywhere dispersed in Case x, whilst in Case xi there were scattered nuclei in the cardiac and middle regions.

In both of these cases the mucous membrane was very thin; in Case xi twenty grains of it carefully dried were digested in ether, and lost 32·8 per cent. in weight. But, as the average amount removed by ether in five cases dying of other diseases was only 12·6 per cent., it is evident that in the cases just narrated considerable amount of fatty degeneration must have taken place.

Eleven grains of solid albumen were digested in an acidulated infusion of the mucous membrane of Case xi for twelve hours, and, although the albumen became translucent at the edges and

softened, it gained one grain in weight. As the average solution of albumen in similar experiments in cases dying of other diseases amounts to four grains, it is plain that the functional activity of the stomach in Case xi had been greatly diminished.

In the next four cases no serious disease of the gastric tubes could be observed by the microscope.

Observation xii.—A woman, æt. 60, died in November, 1864. The mucous membrane of the stomach was remarkably thin. The tubes were distinct and readily separated, but were loaded with granules and fatty epithelium.

Observation xiii.—A woman, æt. 50, died January, 186–. The mucous membrane was very thin, weighing only three and a half drachms. Torulæ existed in great numbers in the contents of the stomach. The tubes could be easily separated, but were chiefly filled with granular matter.

Observation xiv.—A woman, æt. 49, died July, 1863. The stomach was quite undigested, although that of another person who died the same day of another complaint was extensively acted upon by the gastric fluid. The mucous membrane was everywhere very thin, and the blood-vessels were greatly enlarged. The tubes seemed healthy, and contained cells. The duodenum was very granular, and Brunner's glands were much enlarged.

Observation xv.—A woman, æt. 59, died January, 1865. The stomach was deeply congested, and the veins were very large. The tubes could be easily separated, but the gastric cells were large and fatty, breaking down with the least pressure.

In none of these four cases was there any evidence of structural disease in the stomach, excepting in the softness of the gastric cells, and the unusual amount of granular matter; but in Case xiv the duodenum was very granular, and its vessels large and injected. Although three of them presented the usual thinness of the mucous membrane, in one this membrane weighed nearly the average, viz., 720 grains.

In one case ether removed 19·1 per cent. of weight, showing that some amount of fatty degeneration had taken place. In two cases, in which artificial digestion was tried, there was a mean loss of 4·8 grains of albumen; so that, as far as pepsine was concerned, there had been no deficiency.

It will be remarked that the state of the pyloric region has not been mentioned in the foregoing cases. All reference to it has been omitted on account of the frequency with which this part of the stomach presents morbid changes, especially at and after middle life.

But, it may be asked, if disease of this part so constantly exists, apparently without any constitutional affection arising therefrom, are we justified in supposing that a similar condition of the splenic and middle regions is likely to produce more serious results? I think a consideration of the comparative extent and activity of these different regions will enable us to decide the question. In nineteen cases of death from other diseases than cancer I found the average weight of the mucous membrane of the middle and splenic regions to be thirteen drachms, whilst that of the pyloric only amounted to two drachms. In seventeen instances artificial digestion was tried with mucous membrane taken from each region, and the average amount of albumen dissolved by two drachms of the pyloric was only 1·4 grain, whilst by two drachms of the middle and splenic it was 3·1 grains. In bulk, therefore, the pyloric did not constitute one seventh of the whole membrane, and the material of which it was composed had little more than one third of the digestive power of the more active portions of the organ.

The proportion of these parts also varies according to the age of the individual; thus, of five cases who had died under twenty-five years of age the pyloric formed only 11·9 per cent. of the whole mucous membrane, whilst of fourteen above that time of life it amounted to 16·4 per cent. We may, therefore, conclude that when the growth of the body is fully completed the pyloric region normally undergoes some alteration of structure, and thus the anatomical changes so frequently found in it may not effect nutrition.

But is the amount of disease as just described in the splenic and middle regions of the stomach greater than is usual in other complaints? Out of one hundred cases, exclusive of cancer, I found seventeen in which the gastric tubes in these regions had undergone considerable morbid changes, and Dr. H. Jones relates that in fourteen (of which two were persons who had died of cancer) the tubes in these situations were implicated to a great extent. As will be afterwards seen, the diseases in which this most frequently occurs are phthisis and certain heart and liver affections. Of the first of these, according to my own observations, 33 per cent., of the second 50 per cent., and of the last 50 per cent., presented appearances of chronic catarrh in the splenic and middle regions. But as in 75 per cent. of the cases of cancer of the breast the gastric tubes in the middle and splenic regions were extensively diseased, it is evident that this cannot have been a mere accidental complication.[1] In no disease have I seen the secreting tubes so greatly destroyed as in cancer of the breast; in other diseases the changes are usually limited to thickening of the basement membrane, increase of the connective tissue, enlargement of the solitary glands, and alterations in the epithelium; whilst in many of the foregoing cases of cancer all trace of the tubular structure had disappeared from a large extent of surface, or only masses of fatty cells remained to point out its former situation. Again, in cancer of the breast, the mucous membrane is usually atrophied and fatty; but in cases of diseased heart and liver it is thickened, and the epithelial cells are increased in size and number. These morbid changes have, doubtless, resulted from different causes, as will appear more clearly on examining the following table, which contains the causes of death of those persons in whom the tubes were seriously affected in the splenic and middle regions of the stomach, according to the observations of Dr. Jones and myself. I have also added the observations of Dr. Fox, although he includes all cases of chronic catarrh, whether limited to the pyloric region or not:

[1] I have since examined two other cases, in both of which the mucous membrane of the stomach was extensively diseased.

TABLE.

	Dr. Handfield Jones.	Dr. Fox.	Dr. Fenwick.
Tubercle of lungs	2	9	4
Other diseases of lungs	...	3	...
Diseased heart	4	7	8
Diseased heart and phthisis	...	5	...
Diseased liver	2	1	3
Diseased kidney	4	4	...
Drunkenness	...	2	...
Fever	...	2	2
Other diseases	...	3	...
	12	36	17

It is evident from this table that the diseased state of the stomach is usually the result of an obstruction to the circulation of the blood, for in Dr. Jones's cases 66 per cent. in which this condition exists, consisted of diseased lungs, heart and liver; whilst in Dr. Fox's 60 per cent., and in my own 88 per cent., are to be referred to the same cause. But in cancer of the breast there is no mechanical obstacle to the flow of blood through the mucous membrane, and the frequency with which it is diseased must therefore arise from some other circumstance than that usually producing gastritis.

The bulk of the whole mucous membrane of the stomach varies according to the sex; thus, in six females who had died of other causes than cancer, the weight, on the average, was 730 grains; but in twelve males it was 1027 grains. Again, it varies according to age; thus in four males from twenty to twenty-five years of age the average weight was 1130 grains, in eight above that period of life it was only 1008 grains.[1]

As cancer is most frequent in the female, and the liability to it increases with age, it would be interesting if we could ascertain whether the mere loss of bulk observed in

[1] In these cases the mucous membrane was in a moist condition.

three of the cases in which there was no structural disease of the mucous membrane had been sufficient to produce any effect on the general health.

Cancer of the Uterus.

In only three out of twenty-four cases was there any serious change in the mucous membrane of the stomach similar to that described as so often occurring where the disease had affected the breast.

OBSERVATION XVI.—A woman died in August, 1863. One part of the mucous membrane of the middle region showed no remains of gastric tubes, the whole tissue was white, opaque, and loaded with nuclei. In another part the tubes could be distinguished, but were firmly united together, their basement membrane being much thickened and the spaces between and below them loaded with nuclei.

OBSERVATION XVII.—A woman, æt. 30, died February, 1865. A large hard ulceration, but which showed no appearance of cancer, was found in the middle region near the pylorus. In the splenic region the mucous membrane contained only a few bulbs as the remains of gastric tubes; the solitary glands were enlarged, and the whole tissue was loaded with cells and nuclei.

In another case there was a large hard patch of thickened tissue, like the cicatrix of an old ulcer, and the secreting tubes in every part of the organ were much diseased.

In five other cases the anatomical condition was of a different character; the tubes were easily distinguished, but were very soft; they adhered to each other and to the muscular layer by very fine fibres, and they usually contained transparent cells. The blood-vessels were generally congested, and the surface of the membrane was covered by a considerable quantity of mucus. From the fact that inflammation between the gastric tubes usually terminates in other cases in

their adhesion to each other, and from the congestion of the blood-vessels, I suspect the above cases to be of the same nature as those next to be described, excepting that chronic inflammation had taken place.

In sixteen cases the anatomical characters were as follows:— The tubes could be readily separated from each other, but the basement membrane was exceedingly thin; the tubes contained gastric cells, more transparent than usual, breaking down into granular matter with very slight pressure. The mucous membrane was but little diminished in bulk, the average of three cases being 660 grains; it was very pale, but this, I think, is not entirely to be attributed to the discharges produced by the local disease, for in cases in which the connective tissue was increased it did not exist to the same extent. In some instances the tubes seemed bathed in an albuminous fluid; in others the membrane cut as if gelatinous, and in but few instances did spirit or chromic acid harden the membrane as is the case in its normal condition. The chemical composition likewise varied; in one case the softness seemed to be connected with an increased deposition of fat to the extent of 33·5 per cent. of the entire substance; in another there was nearly 50 per cent. of albumen, whilst in a third with a small proportion of fat and albumen there was a large excess of gelatine. It is difficult to estimate the effect these changes had produced upon the function of the organ. As a general rule, post-mortem solution was not common, but I have met with it in a few cases, and chiefly, I think, in those in which the change in the membrane was of a fatty character. In nine cases artificial digestion was tried, and in one 3·7 grains of albumen were dissolved; in the remaining eight, although the albumen was softened, no loss of weight had taken place.

Whether, therefore, we choose to refer the loss of pepsin[1] to an alteration of structure or to the anæmia consequent on

[1] Some of these specimens had been kept for some weeks in spirit, but artificial digestion was readily effected by the mucous membrane of stomachs of other cases than cancer which had been similarly preserved for the same length of time.

the cancer, there can be little doubt that during life digestion must have been imperfectly performed.

As 75 per cent. of the cases of cancer in the breast and only 12 per cent. of the cases of cancer in the uterus presented morbid changes in the mucous membrane of the stomach, it is probable that the nature of the cancer affecting these organs respectively is different. This opinion is strengthened by the fact that I cannot call to mind a single case in which both the breast and the womb were simultaneously diseased.

The remaining cases in which the condition of the stomach was carefully examined are grouped in the following table according to the site of the cancer:

Cancer of other parts than the Breast or Uterus.

	Sex.	Age.	Condition of stomach.
Cancer of tongue	Male	50	Enlarged solitary glands; tubes united, and in places atrophied.
	Male	...	Enlarged solitary glands; tubes united; large perforating ulcer.
	Male	...	Tubes normal.
Cancer of stomach	...	...	Tubes in some places united.
	...	...	Tubes extensively diseased.
Cancer of rectum	Female	54	Tubes united by fine membrane.
	Male	26	Enlarged solitary glands; tubes diseased.
Cancer of groin, penis, and bladder	Male	...	Great thickening of connective tissue, with adhesion of tubes.
	Male	72	Tubes normal.
	Male	50	Tubes normal.
Cancer of glands, bones, skin, and lungs	Male	71	Tubes normal.
	Female	59	Tubes normal.
	Female	...	Tubes normal.
	Male	82	Tubes normal.
	Male	...	Tubes normal; connective tissue increased.
	Female	45	Enlarged solitary glands; adhesion of tubes.
	Male	29	Tubes normal.
	Male	43	Tubes normal.

I have mentioned a case (Observ. xvii) in which a large ulceration had formed in the stomach in a person suffering from cancer of the uterus, and it will be observed that a similar lesion occurred in a patient affected with cancer of the tongue. In both, the connective tissue below the muscular coat was greatly thickened, and this condition was also present in another case of cancer of the bladder, in which although no ulceration had taken place, pus cells were found in and the mucous membrane.

The class in which disease of the stomach has been most rare is that composed of cancerous affections of the skin, bones, and glands.

As the changes in the stomach are most frequent in cancer of the breast, the organ in which scirrhus is most common, and least frequent in cancer of those organs which are especially liable to the attacks of the softer varieties of the disease, it is, I think, probable that the alterations in the gastric tubes will be found to coexist only with the harder forms of malignant growth.

From the similarity of the intestine in structure to the stomach, we might expect to find anatomical changes in it also. In the earlier examinations I unfortunately neglected to investigate the condition of the intestine, but in the last twenty-three subjects, in which I have minutely examined it, I have usually found it diseased.

An increase in the quantity of nuclei in the intestinal villi was observed in Cases x, xi, and xiv. When this form of disease was present the mucous membrane was usually more firm than in its normal condition. The nuclei were very prominent and distinct, there was generally a deficiency of granular matter, and the basement membrane was often thickened. Brunner's glands and the intestinal tubes often presented similar changes, their cells being intermixed with nuclei. In Cases i and ix, and in a case of cancer of the uterus, the villi were exceedingly fibrous, and looked wasted. The fibres were disposed in a direction parallel to the long axis of the villi, and between them were lines of

prominent nuclei. In some instances the ducts of Brunner's glands were greatly thickened and enlarged.

In Case xv, and in two cases of diseased uterus, fatty degeneration of the mucous membrane was present. The appearance of fat was not the result of digestion, for Brunner's glands presented a similar condition. The villi were very thin and transparent, and so soft as to be crushed with the least pressure. Small globules of oil were diffused everywhere, and in many parts the intestinal tubes were either absent or the remains only of their closed ends were visible.

The most common morbid appearance was a dark condition of the villi; indeed in only three cases out of seventeen where no disease of the stomach existed was this change wanting, but where the stomach and villi were loaded with nuclei it did not present itself excepting to a small extent in one case; in two out of the three exceptions the mucous membrane was in a soft fatty condition. To the naked eye the mucous membrane appeared in many cases of a uniform dark hue, whilst in others the colour was more intense, and the villi could be readily distinguished as minute black spots. When examined by the miscroscope the villi were seen to be loaded with dark cells and nuclei, which were in greatest numbers at the free ends, but often extended the whole length. In some instances the villi were universally coloured by dark spots of an irregular form; occasionally they were loaded with a fine, dark, granular matter, and, in two or three instances, I think, I detected minute crystals in their interior.

I need not say that the dark appearance of the villi is not peculiar to cancer; it has been described by other observers than myself. I have met with it chiefly in fever, but as I have never found it in men who have been killed by accident, and once only in upwards of 100 dissections of wild animals, we may assume that it indicates an important alteration in the intestine.

The similarity in the appearances presented by the villi in typhus and cancer is of great interest, tending, as it does, to support the opinion of Rokitansky on the analogy between

typhous deposits and carcinoma. Thus, in his conclusions respecting typhus, he states, "The product of typhus presents in its first, but still more in its later stages of metamorphosis, the greatest analogy with cancerous growths, and more particularly with medullary cancer."

I have often seen oil-globules in villi thus affected, so that, as far as the mere absorption of fat is concerned, their functions are not destroyed. We know, however, that the albumen of the chyle undergoes changes in its passage through the lacteal system; and as many of the softer forms of cancer seem to consist chiefly of this substance, it is, I think, probable that a morbid condition of the villi is connected with functional changes of an important character.

It will be observed that out of fifty-seven cases of cancer the stomach was seriously diseased in twenty-one, or in 37 per cent.; and that in nineteen cases in which the intestines were examined, and in which the gastric tubes were normal, morbid changes were detected in eighteen, so that we have reason to suppose that few cases of cancer die in which serious anatomical change of a non-malignant nature is not present in some part of the digestive canal.

But the important question arises, what relation have these conditions of the mucous membrane to cancer? Are they produced by it? Are both the results of a common cause, or does cancer originate from the disease in the digestive organs? Although I think the information we possess is insufficient to answer these inquiries, a few observations upon them may, perhaps, be useful.

With regard to the first question, it may be remarked that the changes in the stomach are most striking in cases of cancer of the breast, the form of cancer in which operation is most frequently resorted to, and in which, consequently, the local disease has a shorter time to infect the system. In many of the foregoing cases the alterations in the gastric tubes were most advanced when the amount of cancerous disease was small, and confined to the neighbourhood of the breast. When we couple with these considerations the fact that the alterations in the digestive canal are not peculiar to

malignant disease, we can, I think, only conclude that they are not produced by the action of the tumour.

The degeneration of the arteries that so frequently accompanies cancer would seem to show that a common cause is likely to have produced the degeneration, as well as the changes in the digestive tube and the cancer itself. When the nuclei and fibres are developed in the stomach even the smaller arteries of other parts are often found hard and rigid, whilst in cancer of the uterus atheromatous deposits in the aorta are frequently met with; but, on the other hand, fibrous and fatty degeneration of the liver and other organs are of everyday occurrence without the development of cancer, and there seems no reason if the disease in the arteries, stomach, and breast originate from a common cause, why other organs should not be, at the same time, implicated.

In favour of the theory that the changes in the digestive canal act as *predisposing* causes of cancer is the circumstance that the cancerous material appears to consist of an altered form of albumen, and we might, therefore, reasonably suppose that disease in the organs whose office it is to prepare this material would have a tendency to produce an alteration in its qualities. In addition to this is the fact that different parts of the digestive canal seem to be affected in various degrees, according to the organ in which the cancer exists.

But, at whatever conclusion we may arrive, a knowledge of the frequency with which diseases of the digestive organs present themselves in cancer cannot fail to prove useful to the practitioner. He will see in these alterations in the blood-making organs an explanation of the anæmia that so often ushers in and accompanies malignant disease, and he will be encouraged to hope that means may be discovered to arrest the progress of these alterations or obviate their effects upon the system. If further inquiry should teach us that certain varieties of gastric and intestinal disease always coexist with particular forms of cancer, the state of the digestive organs will have to be considered in determining the advisability of operation, as success must depend upon the condition of the internal organs.

In conclusion, I beg to thank the surgeons of the Middlesex Hospital, Messrs. Shaw, De Morgan, Moore, and Nunn, for their liberality in granting me the opportunity of examining the foregoing cases of cancer. I have also to thank Dr. Cayley, Pathologist to the Hospital; and I am especially indebted to Mr. Nunn for the assistance and advice he has afforded me in the course of my inquiries.

DESCRIPTION OF PLATES V AND VI.

PLATE V.

FIG. 1.—*a*. Enlarged solitary glands.
b. Altered mucous membrane.
c. Muscular layer.
d. Remains of an altered gastric tube.

FIG. 2.—Gastric tubes in process of being transformed into fibres.
a. Gastric tubes drawn from their natural perpendicular position.
b. Tubes in state of transformation into fibres.

PLATE VI.

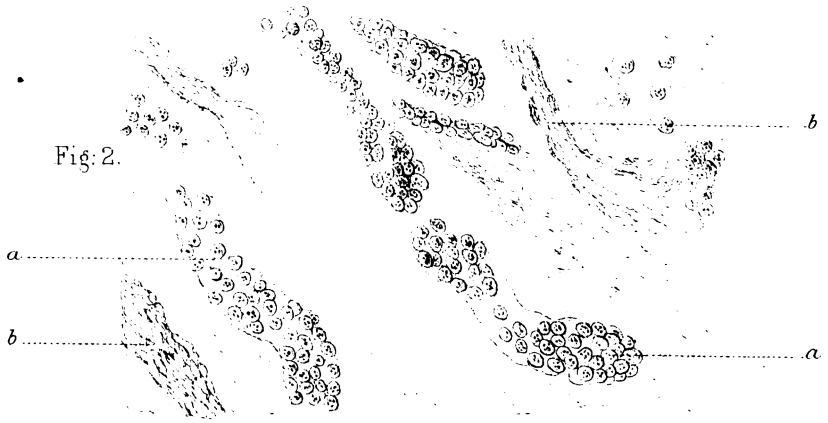
FIG. 1.—Gastric mucous membrane changed into fibres.
a. A few scattered cells.
b. Remains of muscular layer.
c. Elastic fibres.

FIG. 2.—Intestinal villi in cancer.
a. Thickened basement membrane.
b. Nuclei.

FIG. 3.—Ditto. Villus in a state of fatty degeneration.

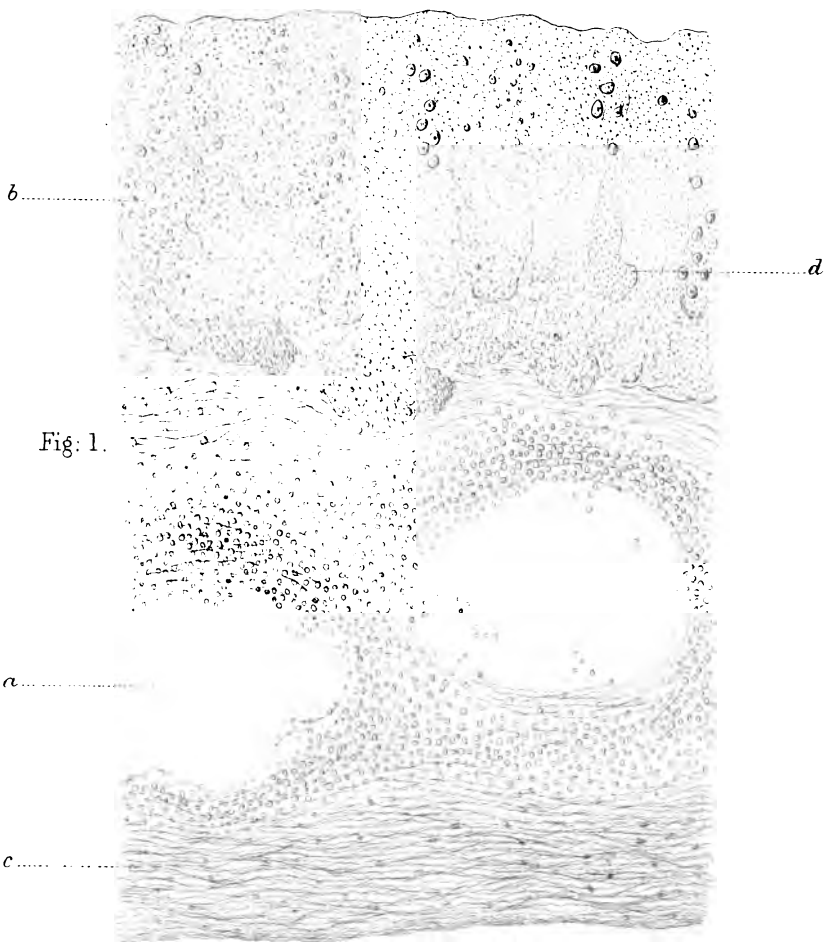
FIG. 4.—Ditto. Villus loaded with pigment.
a. Pigment.
b. Oil-globules.

Fig: 2.



× 125 Diameters.

Fig: 1.



× 125 Diameters.

T. West, del. G.H Ford. W. West, imp.

Plate VI.

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Fig. 1.
x 125 Diameters.

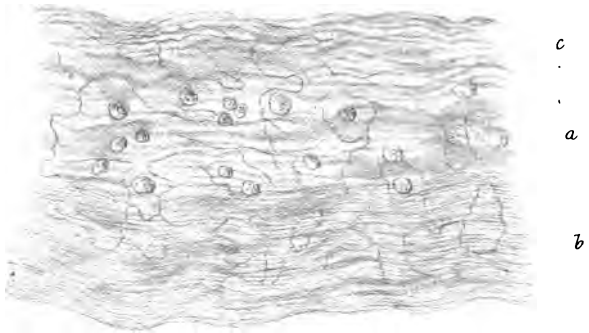

Fig 2.

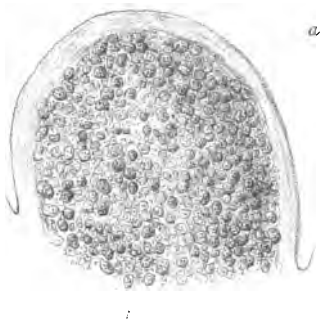

Fig. 3

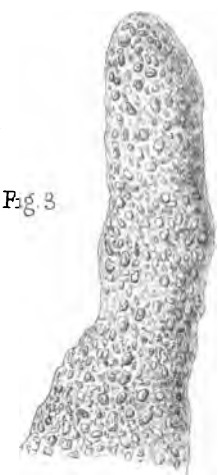

Fig. 4.

Fig. 2, 3 & 4, x 250 Diameters.

G.H. Ford.

W. West imp

FIBROID DEGENERATION OF THE LUNGS.

BY

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COMMUNICATED BY

DR. BARLOW.

Received May 16th.—Read June 27th, 1865.

The subject of degeneration of tissue has of late years attracted considerable attention, and one of the principal circumstances which has led to this result is our better acquaintance with morbid anatomy.

Formerly, if a man apparently in good health was attacked with acute inflammation, and died in consequence, it was thought that the fatal result was altogether owing to the intensity of the disease itself; and the opinion prevailed that death from acute disease alone was not uncommon. But as post-mortem examinations were more frequently resorted to, and as post-mortem appearances were better understood, it was noticed that death from acute disease was very rare in persons whose tissues had not previously undergone degeneration; but that death resulting from the quasi-accidental supervention of acute changes upon chronic degeneration in the tissues was

exceedingly common. Hence the conviction has gradually dawned on the minds of physicians that the kind of, or the success of, treatment depends, not so much on the particular disease itself, as on the healthy or the degenerate condition of the tissues of the patient's body.

I have been led to make these few introductory remarks with the special object of showing that the necessity for the study of the degeneration of the different tissues has not been overlooked; moreover, that a more scientific and a more successful treatment of disease is to be hoped for as the tendency to the degenerative metamorphosis of the different organs of the body is better understood.

Without, however, further trespassing on your time, I will at once proceed to the subject of this paper, and probably I shall best make myself understood by briefly stating what I understand by "fibroid degeneration of the lungs."

On section, part or almost the whole of one or of both lungs was found to be solid, to be increased in weight, to sink in water to be firm, and tough, breaking down under the finger with great difficulty, and in some cases grating when cut with the knife. The cut surface was smooth and non-granular, or at other times markedly granular, and occasionally both appearances were found in the same lung. Not infrequently, on dividing the firm hard mass, the new tissue was found to be perfectly smooth, but closer examination showed that as the indurated part approached the more healthy portion of the lung it was granular, and in parts so much so that it very much resembled a number of minute, hard, cartilaginous semi-translucent tubercles, closely clustered together, which, by their juxtaposition, to use the comparison of Laennec, resembled the eggs of insects. This was especially seen when the lungs were very emphysematous, and consequently some of the best examples were observed in the lungs of old men. This granular appearance was found only in that part of the lung where the connective tissue was increased, and although the granules in one case were isolated, yet each granule was situated amidst augmented interlobular tissue. This granulation the late Dr.

Addison was in the habit of teaching is not due to the presence of the so-called tubercular deposit,[1] but is produced by the intervening new material contracting, compressing, and puckering up one or more air-cells.

The diseased part was dry, and scarcely any fluid could be squeezed out of the consolidated mass. On careful examination it was noticed that the indurated portion of the lung was mapped out in different colours. The connective tissue between the lobules and "lobulets," appeared to be increased, and in consequence was seen unusually distinctly. When a small piece of the diseased lung was placed in water, and a strong light thrown upon it by means of a condenser, it was noticed that the whole of the affected part was divided and subdivided in a ramifying manner by this augmented connective tissue; also placed within and surrounded by this apparently increased tissue, was seen a considerable quantity of dark pigment matter and minute polygonal spaces, containing the air-cells. The whole had a blueish grey, or almost a black colour, or as it has been termed, a "grey granite," or by others, "an iron-grey appearance." Almost the whole of the lung was uniformly affected, or only here and there; as a rule it was not uniformly indurated.

Whilst the solidified portion of the lung seemed the most likely to attract attention, yet on closely examining the pulmonary tissue which had been not yet consolidated, although in the vicinity of the firm fibroid masses, it was noticed that it was pliable and somewhat crepitant, that it did not sink in water, but that the interlobular fibrous tissue was distinctly seen, and evidently increased in quantity. Also it may be observed that, amongst the augmented connective tissue there was a large quantity of very dark pigment matter. Thus the latter appearances would tend to show that mere solidification is not the primary mischief, but that it is preceded by morphological changes in the connective tissue. In proof of this, cases were seen in which there were not any consolidated masses, but the whole or nearly the whole of

[1] Tubercle is here considered to be a lowly organized product made up of granular matter and ill-formed cells.

both lungs was interwoven by a filamentous fibroid tissue, giving the lung the appearance as if it was made up of connective tissue only. (Vide Case No. III, and the post-mortem examination conducted by Dr. Wilks.)

A minor degree of the appearance was not uncommonly seen in the upper portions of the lungs of patients who had died of cirrhosis of the liver, or of granular degeneration of the kidneys, or in the lungs of old people, and it appeared to indicate that the lungs were in an early stage of fibroid degeneration.

This fibroid change appeared to attack as a general rule both lungs, but not always so, and when one lung only, it was most frequently the left. In all the cases which I have yet seen, with the exception of the one given below, it appeared to have commenced in the upper lobes. In one case, the particulars of which will be given, the lower lobes were the seat of change.

This case differed from the description just given, by the entire lower halves of the lungs being uniformly consolidated, firm, and of a reddish-grey colour, and while in the left lung the consolidation was more in patches, nowhere did these patches appear completely isolated from the rest of the solid portions. In the fibroid lungs the bronchial tubes passing through the consolidated tissue were not, as a rule, much dilated, but in some cases the minute tubes towards the surface of the lung appeared to have undergone more or less dilatation, while the congested thickened state of the mucous membrane, and the increased size of the longitudinal fibres, gave evidence of bronchial irritation of some standing.

The sides of the pleura were mostly found to be very firmly adherent, so much so, that it was necessary in some cases to cut the lung out; in other cases the pulmonary pleura over one or even over both lungs, was very much thickened, in one instance to the extent of a quarter of an inch in diameter. In many cases fibroid septa were seen extending into the lung substance from the thickened pleura dividing the lobe of the lung in two halves, and occasionally these septa were

noticed to be thicker and thicker as they approached the pleura.

This fibroid change seemed to take place very slowly, generally extending over months, and in some cases over years.

In two cases it was the only apparent disease present in the body, but more frequently it was associated with allied changes in other organs, such as granular kidneys, granular liver, fibroid degeneration of the heart, and endocardium, and of the pyloric end of the stomach, of the capsule of the spleen, of the skin, and of the bronchial glands.

In the more chronic cases it was not uncommon to find a number of incidental changes in the consolidated portion of the lung.

In some cases there was discovered a cavity in the apices of the lungs, which cavity had very irregular walls, looking as if the lung had been eaten away, or as if the cavity had been artificially produced while tearing out the adherent lung. Besides these, there were some smaller cavities, varying in number, and scattered throughout the indurated portion of the lung; the latter had very thin membranous walls, which were continuous with the lining membrane of one or more bronchial tubes which were seen to be opening into the smaller cavities, thus conveying the impression that they were simply dilated circumscribed portions of the bronchial tubes themselves.

Occasionally an irregular ragged-looking cavity was noticed, partly surrounded by normal lung tissue, and partly by fibroid induration. It was found to contain a most offensive dark-red fluid, and judging from the odour, was thought to have been caused by circumscribed gangrene.

The bronchial glands were generally enlarged, in two cases very much so, and found to have been converted into firm, hard, masses, which like the lung, while being cut, grated under the knife.

Lastly, in the midst of the consolidated parts of the lung, some yellow-looking spots were noticed, which had a soft

consistence, and would apparently come under the designation of the so called "cheesy matter."

Having now described the post-mortem appearances of this form of lung which have appeared to me to indicate fibroid change, I will next give—

The Microscopical Examination.—With the object of endeavouring to throw some light on the nature of the pathological changes that had taken place, sections were taken from different parts of the lungs.

1st. From that part of the organ which apparently had been not yet indurated, although situated in the immediate vicinity of a mass of consolidated pulmonary tissue.

2nd. From that part of the lung which had undergone the process of induration and consolidation.

A section taken from the former situation showed some granular matter, a great number of corpuscles and well formed cells. The corpuscles were mostly spherical or ovoid in shape, with granular contents.

These corpuscles varied in size, as is shown by this diagram. (See Drawing, No. I.)

Although in a great many of these granular corpuscles no nuclei were discovered, yet in many others a minute central nucleus could be seen, especially if magnified about 500 to 700 diameters; under which circumstances many of these granular bodies appeared as well formed cells, each cell having granular contents and central minute nuclei.

The forms of these cells varied from the small spherical nucleated body to the well developed spherical and fusiform cell; besides these, were others which appeared to be in an intermediate stage of development. The magnitude of these cells also differed. (See Drawing No. I.) In one case, the clinical history of which seemed to indicate a comparatively rapid formation of this fibroid change, the cells were large, and the nuclei very distinctly and prominently seen; and the latter of such a size as to approach in their appearance some of the so-called "cancer cells."

While it was difficult to decide from what tissue of the air sac these newly-formed corpuscles and cells had directly

originated, yet it could be clearly determined that the air sacs were not merely filled up by this new cell formation; for these corpuscles and cells were seen to be lying everywhere amidst the elastic fibres forming the walls of the air-cells. (See Drawing, No. II.)

This arrangement of these cellular elements was easily made out by carefully focussing, and the elastic fibres of the air-cells were plainly seen; and by still further adjusting the focus, these corpuscles and cells were noticed lying at different depths amongst the elastic tissue.

In order to demonstrate a further stage of this disease, other sections were made from the consolidated parts of the lung. When besides the corpuscular and cellular elements that I have already described, the air cells were seen to be obscured, and entirely obliterated by a newly formed material which had the appearance of a very fine fibre-like tissue. The linear markings of this new tissue were very distinctly seen when magnified to 125 diameters; but on still further increasing the magnifying power to 750 diameters, the tissue was seen to be made up of very fine, slender, spindle-shaped fibres, which lay side by side with one another. (See Drawings, No. III and IV.) These new fibres appeared in parts to run somewhat irregularly, yet they were mostly observed extending across and completely filling up the air-cells, so much so, that it was difficult to recognise any normal lung structure at all. If, however, the focus was adjusted, the elastic fibres of the air-cells were discovered crossing the new fibre-like tissue in various directions.

In other parts, however, so high was the development of this new tissue, and so complete the filling up of the pulmonary cells, that no trace of the elastic tissue of the lung could be found, until a little of a comparatively strong solution of diluted acetic acid, or a little of a solution of bicarbonate of potash was added, when the new fibre-like tissue swelled up, became translucent, and then the elastic fibres of the air-cells were seen out of shape, shrivelled up, and apparently wasted.

It is not to be supposed that this appearance, denoting that,

the air-cells had been occluded, was equally and distinctly observed in every section, for while in one instance it was so evident that there could not be any doubt of it, in another section taken from almost the same part of the lung, the air-cells and elastic tissue seemed obscured, and almost totally hidden by a quantity of granular matter. This granular matter, however, appeared to have a definite and mostly a linear arrangement. After prolonged and careful observation, it was noticed that the granules were but the contents of very delicate fibres, the walls of which fibres were so translucent that they were with difficulty discovered. (See Drawing, No. I.) Further still, after diluted acetic acid was added to the section having this granular appearance, many of the dark granules disappeared, leaving some fat granules; and a number of ovoid and other shaped nuclei became visible. That these minute ovoid bodies were really nuclei could be definitely shown, by examining a portion of the section which happened to be thinner than the remainder, as for instance, towards the margins, where fine delicate fibres were seen, having ovoid or rounded bodies as nuclei; and further, after soaking a section in carmine solution, the apparent nuclei were observed more deeply coloured than the rest of the new tissue, and the newly formed fibres were seen lying side by side, and in many of the fibres a pink-coloured nucleus. It is scarcely necessary to mention that these nuclei are easily to be distinguished from the nuclei of the pulmonary capillaries.

The granular appearance just described would seem to be due to the fibroid material undergoing degeneration, for it was always very evident after the diseased lung had been kept two or three days. One great difficulty in examining the new tissue arose from the tendency it had to rapidly undergo granular degeneration.

In some situations amongst the newly formed fibres, a number of large nucleated cells were detected, which appeared to be round and other shaped connective tissue cells. In order, however, to see distinctly these connective tissue cells, it was necessary to use an object glass capable of

magnifying about 800 or 900 diameters. (See Drawing, No. V.)

Further, in some parts, this fibre-like tissue was seen arranged in delicate bundles, and in each bundle so highly developed was this new formation, that the individual fibres could be seen without any difficulty.

Further sections were taken from the part of the lung where the new tissue was not smooth on the surface, but granular, especially where the granules were somewhat isolated, and so resembled miliary tubercles. The section from which the Drawing No. VIII was made was taken from the lung of a man aged 80, in which this granular appearance was seen in a very high degree. On pressing a piece of this lung between the fingers, it felt as if it contained a number of minute rounded bodies. These granules were in some parts seen to be distinct from one another, yet each grain-like body was situated amidst the thickened interlobular tissue. The microscope showed that each granule was made up of fibro-cellular elements, and arranged as follows:

A number of spherical corpuscles were lying close together, and in such numbers as almost to fill the entire air-cell. At first sight, corpuscles and some granular matter only were observed; more careful examination, however, with a slight adjustment of the focus, showed these corpuscles to be lying amidst a fibre-like tissue, and many of them seemed to be only the nuclei of delicate looking new fibre cells. The slightest possible adjustment of the focus was sufficient to show this relation. Further, on tracing the corpuscles towards the walls of the air-cells, they were noticed to diminish in numbers, and to be intimately mingled with and at last to be almost entirely lost in the more highly developed fibre-like tissue. (See Drawing, No. VIII.) On moving the field these new fibroid elements were noticed to be more and more highly developed as they gradually approached the interlobular tissue, where the new tissue had the appearance seen in Drawing No. IX. That marked No. X represents a still more advanced stage.

All these three drawings were taken from different parts of one large section, cut by means of a Valentin's knife.

Thus the granular arrangement did not appear to constitute any real difference; and while the microscopic appearance essentially differed from what is seen in ordinary tubercle, yet it agreed with what is described in the filamentous cicatricular or organized tubercle of Gerber (*vide* 'Elements of the General and Minute Anatomy of Man and Mammalia,' by F. Gerber, translated by G. Gulliver, F.R.S., page 308); also with the 'Microscopic Examination of the Granulations, composed of fibro-plastic elements,' described by Mons. Ch. Robin. (See 'Bouchut traité des Maladies des enfants nouveau nés,' 3rd ed., Paris, 1855, p. 347, *et suiv.*)

Thus the granular character appears to be produced by the fibroid elements, growing from the connective tissue of the lung, making their way amongst the elastic fibres, and filling up one or more air-cells; and a further degree of granulation is occasioned by the contraction of the earlier and in consequence more developed new tissue, as explained by Dr. Addison.

Hence it is, that where the fibroid induration is of old standing, the hard fibroid almost black structure is non-granular; and agrees with what Grisolle says with respect to the granulation in chronic pneumonia, "Je crois en outre que l'état granulé sera d'autant moins accuse que la maladie sera plus ancienne." (See Grisolle, 'Pneumonia,' p. 71.)

Although this occlusion and obliteration of the air sacs by this fibrillated tissue was so distinctly seen as not to admit of any doubt as to its nature, yet other conditions of the lung were noticed microscopically, which had a somewhat similar appearance to what had been seen in the fibroid transformation. The appearance most likely to be mistaken for the latter change, is that of a piece of collapsed lung in which the elastic tissue has wasted, shrunk up, and in consequence the elastic fibres lie side by side.

The following characters will, however, serve to distinguish the elastic fibres of collapsed air-cells from the fibre-like tissue of fibroid degeneration.

The fibres of the elastic tissue appear longer, and each fibre can be traced to some distance. The outline of the elastic fibre is darker, more distinctly and more sharply defined, and its centre is clear and bright.

The ends of the elastic fibres are seen curled up, and the fibres themselves arranged more or less according to the shape of the air-cells (see Drawing, No. VI); whereas in the fibroid tissue the margins of the fibres are indistinctly seen and imperfectly defined, the light is not readily refracted through their centres, and they cannot be traced any distance.

This microscopical examination led to the conclusion—

1. That there had been a formation of new tissue elements, and that these were such as are usually considered to represent newly formed connective or fibroid tissue.

2. That the newly formed fibroid tissue had invaded and in some parts destroyed the normal lung tissue, or in other words, the lung had undergone fibroid degeneration.

3. That these fibroid elements were found to be most highly developed in the immediate vicinity of the connective tissue surrounding the minute bronchial tubes, lobules, and lobulets, and also in the vicinity of the thickened pleura, which would appear to show that the cell formation had commenced in the connective tissue of the lung, and that it had extended in every direction amidst the elastic fibres of the air cells, until the latter cells were completely filled up and obliterated.

In my description of the appearance of these fibroid lungs, as seen during a post-mortem examination, it has been stated that amidst the grey fibroid induration there were a few "yellow" or "cheesy looking" spots, and the same in the bronchial glands. In connection with these the question arises, what is the nature or the origin of this yellow or so-called "cheesy matter?"

It is well known that since the time of that illustrious pathologist Laennec, such yellow deposits or formations have been by many regarded as yellow tubercles, and the grey

matter around them as tubercular infiltration, whilst other physicians have considered them to be "grey induration," the consequence of so-called "chronic pneumonia."

I would, however, call attention to some facts which have led me to think that such explanation is not satisfactory.

The study of pathological histology has shown that, as fibroid and other new formations grow, the new growth is supplied with nutritive fluid from the circulation of the healthy tissues, in the midst of which it lies. But as the centre and earliest formed portion of the diseased growth becomes further and further removed from the healthy part of the organ, it is exposed to suffer from irregular nutrition, and in consequence is liable to waste, and undergo degeneration; suffering the same kind of degenerative metamorphoses as accompany atrophies in general, that is, the wasted tissue is replaced either by fatty, by pigmentary, or by calcareous matter.

Experience also has shown that cancerous tumours undergo this atrophy, and become yellow and "cheesy" looking in their centres; also, that fibrous tumours of the uterus and of other organs undergo a similar change. Further, that fibroid syphilitic tumours in the liver, and circumscribed fibroid deposits in the cortical substance of the kidney do the same.

If new formations are liable to this kind of transformation in different organs, are they not equally liable to the same transformation in the lungs?

In fact, such changes do occur, even in the lungs, for cancer of the lung has been found to be yellow and cheesy-looking, and the same with syphilitic fibroid growths in the lungs; moreover, it is far from uncommon to find in the lungs, in the lower as well as in the upper lobes, the remains of lobular pneumonia which have undergone fatty degeneration, and are cheesy-looking in the centre.

The microscope shows the yellow cheesy-looking matter to consist of dark granules, of a still larger quantity of granules having bright sparkling centres, and of irregularly

shaped apparently shrivelled wasted cells, filled with what are usually considered to be fat-granules.

These yellow masses, therefore, appear to be the result of degenerative metamorphoses in the new tissue of the fibroid induration, which degeneration is due to imperfect nutrition.

The new fibroid tissue appears to vary very much in its power of resisting this tendency to degenerate, for whereas in some cases no cheesy looking spots were discovered, in others the yellow matter was abundant.

The same reasoning as is applicable to the degenerative changes of this material in the lungs, is also applicable to the same in the bronchial glands.

The cavities occasionally found at the apices of the fibroid lungs were probably the result of atrophy, of degeneration, and of subsequent softening of the fibroid matter. And for the reason that the apices of the lungs are most liable to suffer from impaired nutrition, so any new formation situated in these parts would be more likely to suffer from the want of nutritive fluid than if situated in other better nourished parts of the lung.

The cavities arising from the dilated tubes may be simply globular distensions of the bronchial tube, or as Dr. Gairdner has suggested, be due to ulceration of the bronchial mucous membrane, the resulting cavity being afterwards lined with a new thin membrane.

The formation of circumscribed gangrenous cavities in the lung has been long since called attention to by Drs. Graves, Andral, and Stokes.

The clinical histories of the following three cases will illustrate some of the leading characters of this disease.

The first case was that of a man, æt. 26, a labourer, who was admitted into Guy's Hospital, October 12th, 1864, under the care of Dr. Barlow, who has most kindly allowed me to make use of the case.

The patient was a man of middle height; and had a well developed muscular and bony system, light brown hair, grey eyes, regular features, and sound regular teeth. His skin

was fair, and, when he was well, he was said to have had a good colour.

He stated that he had always enjoyed good health excepting an occasional cold in the winter, and that all his relations were healthy strong people, excepting a sister, who had died of "galloping consumption."

Four months ago his appetite began to fail, and about the same time a cough came on. He remained at his work as a labourer for a month longer, when he was compelled to give up on account of his increasing weakness. During the last three months he had got weaker and weaker, and the cough had continued the same. About three weeks ago his phlegm was slightly streaked with blood.

On admission the physical signs were as follow:—Posteriorly all over the right side, the respiration was feebler than over the left, and there was greater dulness on percussion over the right than over the left side; over the left base, however, percussion resonance appeared somewhat diminished. Vocal resonance markedly increased over the right base. Heart sounds clear and sharp; pulse small and compressible. His skin was not particularly hot; his appetite varied. During the first ten days that he was in the hospital his cough became easier, and he seemed to gain strength.

October 21st.—Immediately under the right clavicle scarcely any respiration could be heard, and there was distant crepitation. Posteriorly over the right apex tubular breathing, with moist sounds and whispering bronchophony were audible, and there was tubular breathing at the right base. There was crepitation all down the left side.

25th.—Respiration laboured, 38 per minute; friction sound heard over the right base with each respiration; pulse 140, very small and feeble; he was bathed in perspiration, and at 2.30 he died.

Post-mortem showed signs of recent pleurisy, especially over the right lung; no firm adhesions anywhere. In the pleuræ, over the lower lobes of both lungs, were a number of minute, hard, grey bodies, the size of split peas.

On section, the upper lobe of the right lung was of a dark-

red colour, and the interlobular tissue was seen more distinctly than usual, as if increased. A very small quantity of fluid exuded from the divided surface, and this part of the lung did not break down under the finger. The whole of the lower half of the right lung was solid, firm, and somewhat tough. From its density and firmness it offered a certain amount of resistance to the knife. It did not readily break down under pressure of the finger, and this, with the increased toughness, distinguished it from ordinary hepatized lung. It did not grate while being cut, but it was of a greyish-red colour. It sank in water, and exuded scarcely any fluid when squeezed between the fingers.

The left lung was in a similar condition, except that the consolidation was arranged more in patches.

The bronchial tubes were much congested, but did not appear dilated. The bronchial glands were very much enlarged, some of them being as large as a walnut, and several of them were so intimately united together by a very firm greyish-red looking material, that it was only with great care and perseverance that the individual gland could be dissected out. The enlarged glands were seen accompanying the smaller divisions of the bronchial tubes, and they were so numerous and so large, that when those of one lung alone had been dissected out, they filled an ordinary plate. On dividing some of the glands, the following changes were noticed:—They were all very firm and tough, and increased in density, and many appeared as if they had been converted into fibrous tissue. Others appeared to have undergone fatty degeneration, and were yellow and decidedly softer than the rest, yet all around this yellow matter was the hardened gland tissue. A few had undergone a still further change, and were beginning to be white and chalky. The pericardium and substance of the heart appeared normal; the valves also were healthy, but in the left ventricle one of the carneæ columnæ was puckered and contracted in two or three parts. On section, this part showed a greyish-white fibrous appearance, as if it had undergone fibroid change. The spleen was very large, weighed fourteen ounces, and was very firm.

Liver appeared normal. Kidneys large, very firm, and tough. Intestines normal, and did not show the slightest sign of ulceration, or of tubercular deposit. The mesenteric glands were not enlarged. Testicles appeared healthy.

The *microscopical examination* showed the air-cells to be filled with a quantity of granular matter, minute granular bodies resembling "free nuclei," large granular corpuscles, and well-formed nucleated cells, some of which were spherical, some ovoid, and others elongated.

Besides these were a great number of well-formed fusiform cells lying amongst the lung tissue, and also seen floating about the field. Most of the latter cells had distinct and very visible nuclei. Lastly, there was some of the ciliated epithelium of the air passages.

The microscopical examination of the bronchial glands showed somewhat similar appearances, with the exception of the fusiform cells.

To sum up the result of the microscopical examination.

The vesicular and intervesicular tissues contained the elements of newly formed tissue in the shape of granular matter, minute spherical granular cells, with visible nuclei, *i. e.*, the round connective tissue cells, and fusiform granular cells with central nuclei.

It will be admitted that this microscopical examination resulted in showing that new tissue elements had been formed, and that such elements were in process of formation when death took place. Further, that the lung was being gradually invaded and destroyed by such cell elements as are usually considered to represent fibroid formation, or, in other words, that it was undergoing fibroid transformation.

Unfortunately while the microscope is capable of pointing out certain tissue elements it does not indicate the nature of the change by which these forms were produced. Therefore, the question will at once occur, were the changes that had taken place in this lung the consequence of an attack of acute pneumonia? And in what respect did they differ from ordinary hepatization?

Some physicians have described a form of induration of

the lung which they have considered to be the result of acute inflammation, and to which they have given the name of "red induration." They believe that acute pneumonia may reach the stage of red hepatization, and then, instead of undergoing the ordinary process of resolution, the hepatized mass may acquire increased consistence, become tougher, and thus give rise to the so-called "red induration."

Lebert, in his work entitled 'Physiologie Pathologique,' gives the case of a man who was considered to have died of pneumonia after a month's illness. The post-mortem showed that the inferior and middle lobes of the right lung were completely hepatized, but with a decidedly increased consistence, just the opposite of ordinary hepatization. The microscope showed granular matter and some fusiform cells.

The late Sir John Forbes recorded a case of acute pneumonia, which had become chronic, and in that case also the lung had increased consistence.

Moreover, there are other facts tending to show that pneumonia, especially lobular pneumonia, does leave behind it certain forms of induration of the lung, a fact to which the late Dr. Addison drew particular attention. Nevertheless, there is the evidence of so many careful observers to show, that it is an exceedingly rare occurrence for ordinary acute pneumonia to pass into chronic.

If this red induration is simply due to acute pneumonia having become chronic, why is not this particular induration more common?

It might be argued that this is owing to some peculiarity in the constitution of the patient; but that would be an admission that it is not the result of ordinary acute pneumonia, but of pneumonia occurring under special circumstances, and the conclusion is inevitable that further research is necessary before it can be admitted that an ordinary hepatized lung ever does become thus indurated and converted into fibroid tissue.

But in answer to the question, were the changes in the lung of this patient the result of acute pneumonia? It is to be remarked, that although the physical signs and general

symptoms that were noticed shortly before death, might lead to the conclusion that the patient was suffering from pneumonia, yet when it is remembered that his illness had commenced four months before, and had come on so gradually that he continued at his ordinary work during the first month of the four; that there was no history of acute inflammation of the lungs previous to his admission into the hospital, but that, on the contrary, the medical gentlemen who attended him during the first part of his illness thought he was suffering from phthisis complicated with Addison's disease; that the post-mortem examination showed none of the ordinary appearance of acute pneumonia; that the microscope proved the nature of the change to be that of fibroid formation; and lastly, that there was fibroid change in the bronchial glands, and to a certain extent in the endocardium, it appears to be exceedingly difficult to prove that the changes in the lungs were due to inflammation alone, and not to a process resembling an acute fibroid transformation—a species of metamorphosis dependent upon some general cause, acting on more than one organ of the body. In support of this view, I would mention that in Lebert's case, which bears strongly upon this question it is stated that the patient had been admitted for pneumonia, and that the illness lasted only a month.

I have been induced to dwell particularly on this case, not so much because it is exceedingly rare, nor to establish my own views, but in hopes that it might throw some light on the whole subject of "red induration" and of comparatively acute fibroid transformation of tissue.

The next two cases are types of a much more common class of fibroid disease of the lung, and are good examples of the kind of pathological change to which I now more especially desire to ask your attention.

August 15, 1864.—I was consulted by M. G. J—, æt. 43, by occupation a straw-bonnet blocker.

He stated, that he had always enjoyed the best of health until about ten or twelve years ago, when he suffered from what

he termed "weakness." After a month's rest, however, he recovered to such an extent as to be able to resume work. At the same time, although he had gone to work, he had never been the same man since that illness.

He had always worked hard, and had had a good deal of anxiety. He had lived freely, drinking chiefly "ale, gin, or brandy and water," but he had not been in the habit of getting intoxicated, nor had he neglected his work a single day from the effects of drink. He was often in the habit of vomiting in the morning. Five years ago he had a mild attack of gout. During the last three years he had been declining gradually, but surely, suffering a great deal from want of appetite, flatulence, and acidity, and from occasional vomiting; which symptoms his medical attendant referred to his liver, which was considered to be very much enlarged.

For the last three or four years he had been troubled with shortness of breath, especially on exertion, accompained with troublesome wheezing, and had suffered with cough every winter, but the shortness of breath and cough had got very much worse during the last six months. About two years ago he got very stout about the belly, and at the same time began to feel less inclination for exertion. The chief things he had now to complain of were want of appetite, a distressing cough, and shortness of breath, especially when walking quickly or during any undue exertion; and of great loss of flesh about the arms and legs, especially during the last twelve months.

In bodily conformation his figure was tall, and well built, he had good-sized bones, and a well-developed chest; the skin was fair, and not particulary thin.

Physiognomical appearances.—He had a good-sized head, and light-brown hair, which was rather thick, and had begun to turn gray, but not much so; his eyes were blue, and the alæ nasi thin; the upper lip was rather thick; his teeth were good, regular, and sound, excepting that the molars and the incisors of the lower jaw were somewhat worn down; his chin was rounded; his complexion and conjunctivæ rather yellow.

He further stated, that when younger he was remarkably vigorous and strong, and thought himself that he had an excellent constitution.

Family history.—Both parents had enjoyed good health. Father died of cholera, aged sixty-five; mother died aged sixty, cause unknown. Has two brothers now living, also one sister, who is a healthy, well-built woman. Two brothers died of scarlet fever when children.

Physical signs.—Forced respiration showed imperfect expansion of the thorax, and the visible expiratory movements were unusually prolonged; during inspiration the supra-clavicular spaces sank in a good deal. Percussion showed diminished resonance under both clavicles, but more especially under the left; the percussion note over the right sub-clavicular region had a "boxy quality." Tubular inspiration, and no vesicular rustle was heard. During forced or normal respiration the expiratory sound was greatly prolonged, and was heard so all over the chest; the vocal resonance was decidedly increased over the left apex, and there were diffuse and large crepitations all over the chest. Heart sounds:—systole short, sharp, and high pitched; rhythm regular; apex beat seen in the epigastrium. Liver:—dulness extended upwards, and its edge was very distinctly felt as low down as the umbilicus; spleen could not be felt; urine albuminous.

On October 27th he complained of severe pain under the right nipple, especially on coughing—this he had felt since the day before. Percussion over the right base showed no diminished resonance; but a well-marked pleuritic rub was heard with inspiration and expiration. Subcrepitant râles were heard over the left subclavicular region. He was very thirsty; the skin was rather hot; the cough very troublesome, and the breath remarkably short. He observed that his breath was "so wheezy" that he felt as if he would choke; urine still very albuminous.

The pain somewhat abated, but on the fifth day he complained of slight hiccup.

For fourteen days he continued much the same, one day

a little better, another day worse. The physical signs appeared to indicate that there was disorganization of the left, and a partial consolidation of the right apex; and acute pleurisy all over the right side. He gradually got weaker and weaker, rapidly losing the little flesh he had, especially about the limbs, although the abdomen remained the same.

November 14th.—Complained sadly of his breath, also that his expectoration smelt very offensive, and to me, and to others, it had a very offensive odour. This lasted two days only, but on the 17th he appeared very ill; his pulse was very quick and feeble, and his tongue dry, and covered with thick, yellow fur, and red at the edges, and he complained of great weakness and depression.

About 3 o'clock next morning he awoke and complained that something was the matter with his chest; he partly raised himself up in bed, when a quantity of blood gushed out of his mouth; he endeavoured again to get out of bed, when another gush of blood took place, and he fell back dead.

Post-mortem, thirty-six hours after death:

Body very much wasted about the limbs, but not about the abdomen. Feet slightly œdematous. Cartilages of the ribs were not ossified. Pleuræ over right lung united by recent adhesions, and between the adhesions were a few ounces of straw-coloured fluid, with a great quantity of soft lymph over the base of the right lung and on the surface of the diaphragm. The left pleuræ were united by very firm adhesions.

The lungs—the upper lobes of which were very firm—were solid, and contained a great quantity of pigment; on section they grated under the knife, and the cut surfaces showed a smooth and non-granular appearance; the connective tissue apparently was much increased; the whole was of a gray granite colour. In the upper part of the lower lobe of the left lung was a small cavity about the size of a large walnut, which contained a quantity of dark-red fluid, having a very offensive odour; careful examination showed that it had ragged walls, and was not lined by any membrane. Opening

into this cavity was a small branch of the pulmonary artery, into the open mouth of which vessel a fine probe was easily passed. The rest of the lung appeared normal. The bronchial tubes were much congested, but not apparently dilated. The bronchial glands were enlarged, and contained a quantity of black pigment. The heart appeared normal. The liver was much enlarged, granular on the surface, of a pale yellow colour, and looked as if it was fatty; at the same time it was tough, torn with difficulty, and cut very firmly. The kidneys were enlarged, congested, and beginning to be granular. The spleen was congested; the intestines appeared normal, and the most careful examination did not detect the least ulceration or any tubercular deposit.

The third case was that of—

J. S—, æt. 50, a labourer, who was admitted into Guy's Hospital, August, 1864, under the care of Dr. Rees, who has kindly allowed me to refer to this patient.

He was a man of middle height, and had good-sized bones and well-developed muscles. His hair was thick, and beginning to turn gray. His features were regular, and his eyes light gray. His alæ nasi were thin, but his upper lip was somewhat thick. His teeth were very regular and sound. His skin was fair and rather thick. He was much wasted, and complained of a troublesome cough and a difficulty of breathing.

He stated that he had enjoyed very good health, and had always had a regular and good appetite, but had drunk freely of spirits for many years. He had been always ready for his work—in fact, he had never had a day's illness up to seven years ago, when he began to suffer with cough and shortness of breath. The cough returned every winter; but, nevertheless, he still continued to do his work, and thought himself "stout and hearty." About two years ago he began to lose flesh, and ever afterwards continued to do so, gradually getting thinner and thinner. His cough and shortness of breath had also got worse and worse. He had never spat blood, but had usually expectorated a quantity of frothy looking phlegm.

Physical signs.—There was diminished resonance on percussion over both apices, but more especially over the left, but nowhere was there very great dulness; the vocal resonance was increased over the left apex in particular. Tubular inspiration and crepitation were heard over the left apex. The expiratory murmur was much prolonged all over the chest, and large crepitation was heard over both bases. The heart's sounds were feeble, but otherwise normal. There was no albumen in the urine.

With the object of saving time it will probably be sufficient to say that this patient remained in the hospital some months, during which time he had several attacks of acute bronchitis, which more than once rendered it doubtful if he would survive. He did so, however, and even recovered to such an extent that he confidently expressed his belief that he would soon be able to go out of the hospital. Yet all this time he continued to lose flesh, and ultimately succumbed to an attack of capillary bronchitis.

The autopsy was conducted by Dr. Wilks, and is reported in 'Guy's Hospital Post-Mortem Records,' as follows:

Lungs both adherent. Emphysematous bullæ at the apices; in a few other parts the apices were puckered. Sections of the lungs showed that they were uniformly invaded by a tough fibre-tissue which had destroyed the natural structure and rendered them partially airless, and very hard. There were no circumscribed masses of hard tissue, as is sometimes seen, but the pulmonary texture appeared invaded in all parts; thus the natural aspect was lost, being striated or interwoven with fibrous filaments. The bronchial tubes were filled with purulent mucus, and the minute branches appeared dilated. Heart appeared sound. Intestines and cæcum healthy. Liver healthy. Kidneys apparently healthy, though small.

I will now proceed to notice very briefly some of the most important points in the natural history of this disease.

I have already stated, more than once, that the sufferers from this affection were well-built persons, and appeared to

have been muscular, well-nourished men. They said that when in good health they had a healthy complexion. Their teeth were regular, sound, and firm, even at middle and advanced life, characteristically differing from the early and much decayed teeth of strumous persons. Their hair was abundant and their skin inclined to be thick. They all stated that they had been very strong men, and remarkably healthy previous to their present illness.

The disease appears to be much more common in males than in females, and to occur most commonly during middle and advanced age, although it does occur at a much earlier period of life. It seems to be slow in its progress, not a few cases extending over months, and some over years. In by far the majority of the cases in which I have seen the diagnosis confirmed by post-mortem examination, there has been a history of great intemperance, especially of long-continued and of excessive spirit drinking.

In the record of the last case it was stated that the patient had suffered with cough and shortness of breath every winter for some years. Such a history was very frequently given. A few cases that we have had an opportunity of watching for three or four years, and one case for seven years, presented marked physical signs of vesicular emphysema and of chronic bronchitis, which signs preceded any symptoms of fibroid induration of the lungs. Again, while the general symptoms were such as indicated vesicular emphysema and bronchitis only, there was not any marked loss of flesh; in fact, the patients were often stout; when, however, the physical signs denoted induration of the apices of the lungs, the patients wasted gradually, and, further, when disorganization appeared to have commenced they wasted rapidly.

The disease, however, did not appear in every instance to be the sequel of old-standing bronchitis and emphysema only, for some of the patients attributed the commencement of their illness to what appeared to have been an attack of pleurisy. This was the case especially in one man, a patient of Dr. Gull's, who died in Guy's Hospital of fibroid degeneration of

the lung. He had been in the same hospital, and under the same physician, with symptoms of acute pleurisy, as far back as eight years previous to his death, and he distinctly stated that, "although he appeared at first to recover, yet he had never been well since the date of the inflammation of his lung."

Clinical experience would seem to show that patients of this class are particularly liable to attacks of pleurisy; and judging from the statements so frequently and so confidently repeated, that they had been well, or rather that they were able to work as usual, before they had the inflammation of the pleura, but that they had got worse and worse ever since, it might be thought that this pleuritic inflammation had been the cause of the fibroid disease of the lung. This opinion would appear to be strengthened by the fact before noticed, that the pleura over the fibroid portion of the lung has been not infrequently found very much thickened; also that fibrous bands have been noticed extending from the increased pleura into the lung.

While it is not to be denied that we do see cases in which one lung alone is affected by this disease, and the pleura over this one lung is a quarter or even half an inch thick, while the opposite lung is healthy or merely hypertrophied; also other cases in which both lungs are diseased, the one indurated and contracted, with its pleura of greatly increased consistence, the other lung undergoing fibroid degeneration without any thickening of its pleura; it is not to be forgotten that in many of the cases the pleura has been found to be not greatly thickened; and in some examples of well-marked "fibroid degeneration" of the lungs the pleura was not at all affected; or, if at all, was merely covered with a thin layer of soft recent lymph. Moreover, isolated patches of this fibroid matter have been noticed in the substance of the lung, having no connection whatever with the pleura; and the same fibroid disease, evidently in an incipient stage of development, without any signs of past or of recent pleurisy, has been discovered in the apices of the lungs of men who had been accidentally killed. Bearing all these things in mind, is there not good evidence to show, that pleurisy is not

essential to the development of this fibroid disease of the lung?

Again, in the case in which one lung alone was affected, and its pleura greatly thickened, the microscope showed the air-cells completely filled up by a new fibroid tissue; hence it is probable that the diminished bulk of the lung was due, not to simple compression and consolidation of the pulmonary tissue by the contraction of the thickened pleura, but to the contraction of this new fibroid material.

Taking all the above facts into consideration, it appears that while there is good evidence to show that inflammation of the pleura may excite the lung to undergo fibroid degeneration, yet that there is still further evidence to prove that the lungs may suffer this fibroid transformation quite independently of an attack of pleurisy.

Moreover, considering the clinical history, the habits of the patient, and the associated fibroid changes in different organs of the same body, it would appear that fibroid transformation of healthy pulmonary tissue is not induced by inflammation of the pleura, but may be excited by acute changes set up in the pleura acting upon the connective tissue of a lung, which impaired nutrition has rendered liable to suffer from degenerative changes.

Instead of inflammation of the pleura, profuse hæmoptysis appeared in two cases to have ushered in this fibroid affection.

Another interesting point in the general symptoms was that the activity of the digestive organs was often very little impaired; the appetite remained good and regular, unless there was some complication, such as jaundice, anorexia from excessive spirit drinking, or, as in one case, the existence of so called "scirrhous pylorus."

A still more noticeable fact was the absence of signs of intestinal disease; for although, in a few cases, more or less looseness of the bowels was observed a few days before death, yet the most careful examination of the intestines failed to discover any tubercular deposit, or any tubercular ulceration of the mucous membrane of the small intestine.

This is the more remarkable when it is remembered that

some of the patients had well-marked breaking down of their lungs, and that they had been confined to their beds some weeks.

Knowing that when there is very extensive degeneration of any tissue, there is little, if any, hope of regeneration, it is very important to discover what is the immediate cause of death. By means of such discovery, although we may not be able to cure our patient, we may yet be able to ward off the fatal result.

In the majority of the cases the immediate cause of death appeared to be capillary bronchitis; acute pleurisy or acute pleuro-pneumonia seemed to be the next most frequent cause, and in one case, the particulars of which I have already given, dissolution was due to profuse hæmoptysis, the consequence of a circumscribed gangrene of the lung.

From the description given, this class of cases will have been recognised as one usually considered under the head of phthisis; and remembering the great talents and vast amount of industry bestowed on the study of the latter disease, it would have been next to impossible that the leading clinical facts connected with this fibroid affection should have been overlooked. Accordingly, some physicians have already described its leading symptoms, but have not considered them sufficiently distinctive to justify them in classing it under any other head than "phthisis;" and they have regarded it, like the latter disease, associated with or dependent upon a scrofulous condition of the constitution.

I would, however, beg to offer the following reasons for not regarding this fibroid degeneration of the lungs as due to "scrofula."

1. The microscopic examination of the diseased parts of the lung showed well-formed cells, and the cellular elements in various stages of development were being gradually converted into highly organized fibroid tissue, strikingly different from the granules and imperfectly formed cells of lowly organized scrofulous matter.

2. The yellow cheesy looking matter discovered amongst this fibroid formation in the lungs might be considered as

indicative of "scrofula," yet it is not to be forgotten that the cheesy substance is seen in new morbid tissue which is believed not to be of scrofulous origin, and that it is difficult to distinguish the imperfectly formed cells of strumous deposit from the shrivelled, wasted cells of other kinds of newly formed material that has undergone atrophy and fatty change.

3. The well-developed skeleton, the highly developed muscular system, and the history of general good health and strength, which characterise these cases, are just the opposite of what is seen in persons having the scrofulous constitution.

4. There were no tubercles in the lungs, unless the granular fibroid tissue is considered to be tubercle, and it is undoubtedly different from ordinary tubercle in the lungs—no trace of tubercle in any other organ of the body, and no signs of scrofula in the lymphatic glands.

5. In twenty cases that I have seen, the most careful examination of the intestine showed no tubercular disease of the intestinal mucous membrane, and in eleven cases which appeared to have been examples of this fibroid disease, the records of which I have found in 'Guy's Hospital Post-mortem Reports,' it is distinctly stated that there was no ulceration, and that the intestines seemed quite healthy.

6. The tissue which appears to be primarily affected is essentially different in fibroid and strumous disease; for whereas the fibroid transformation seems to commence in the fibrous and sero-fibrous tissue, the so-called scrofulous disease seems to commence in the mucous tissue.

It is not, however, to be supposed from the above that we may never meet with signs of scrofulous changes in the lymphatic glands of those persons who have fibroid induration of the lungs and of other organs; for a little consideration will show that in a patient of the scrofulous diathesis, whose lymphatic glands may show the effects of scrofulous disease, the habits of life or other circumstances may have been antagonistic to the further progress of scrofulous disease, but favorable to and capable of developing fibroid disease.

Recalling to mind that in the majority of cases these fibroid changes in the lungs had been associated with similar changes in the liver, in the capsule of the spleen, in the kidneys, in the fibrous tissue of the heart, in the endocardium, and in the peritoneum, it appears to me difficult to come to any other conclusion than that these pathological metamorphoses are all the result of some general influences. I am therefore led to agree with Dr. Handfield Jones, that these fibroid changes are due to a general "fibroid disease."

Although the coexistence of a number of morbid affections of a similar nature in different organs of the same body may warrant the conclusion that they all result from a constitutional disease, yet we are not justified in considering such morbid changes as dependent upon any hereditary tendency, or, in other words, upon any particular form of diathesis; for it is quite evident that these changes may be all due to an incidental cause, such as intemperance.

When, however, we find besides this general fibroid transformation, that all the patients have had nearly the same, if not actually the same, conformation of body; that in consequence of certain corporeal characteristics, the type of person liable to this disease can be recognised, even during apparent health, and that by the aid of certain features of the body I have been enabled to decide that a particular consolidation of the lung was due to fibroid, and not to tubercular changes—an opinion confirmed by post-mortem examination—there appears to be good reason for believing that such patients have an hereditary tendency to this fibroid degeneration, or, in other words, have "fibroid diathesis."

In order that this diathesis should be developed into the fibroid disease, an immediate exciting cause is necessary, which in most of these patients appears to have been excessive spirit drinking. In four cases, however, there was no such history, but the patients were very old men; which might suggest to us that, while the abuse of alcohol brought about pathological fibroid degeneration, mature old age, in contradistinction to premature old age, brought about physiological fibroid degeneration.

One more question still remains:—Is it possible to diagnose during life this fibroid from ordinary strumous phthisis?

In some cases I have found it a matter of considerable difficulty, but in the majority such a diagnosis can be easily made, though it is impossible to distinguish between the two forms of disease by the stethoscopic examination alone; for physical signs may indicate diminished functional activity, consolidation, or disorganization of the lung, yet they do not enable us to infer the nature of the pathological change to which such consolidation is due.

The following observations will, however, be found of use in determining this question. In fibroid disease of the lung there are—

1. Physical signs of vesicular emphysema and bronchitis, and conjoined with these of induration of the apices of the lungs, such as diminished resonance on percussion, tubular inspiration, and prolonged expiration, with increased vocal resonance.

2. The build of the patient is such as I have already described it, and there is a history of good health and of a strong constitution previous to his present illness.

3. A history of winter cough and shortness of breath extends over some years, or there is a history of an attack of inflammation of the chest, viz., of pleurisy, of pleuro-pneumonia, and of impaired health ever since.

4. Albuminuria and other signs of granular kidney exist, or there are signs of cirrhosis of the liver, or of fibroid changes in other organs.

5. There is an absence of general symptoms indicating ulceration of the intestines.

6. There is a history of intemperance, especially of excessive spirit drinking.

7. The disease is much more common in males than in females, and during middle and advanced age than during early life.

I will now sum up the leading points of the paper as follows:

1. That there is a form of induration of the lung, which

the microscope shows to be made up of such elements as are considered to denote fibroid changes; that the newly formed fibroid tissue invades the lung in every direction; that this new tissue reaches such a stage of development as to appear as well-formed fibre-like tissue; and that the pulmonary tissue is obliterated and in many parts completely destroyed by this fibroid tissue, or, in other words, the lung undergoes "fibroid degeneration."

2. That this fibroid change is most frequently seen associated with fibroid degeneration in the kidneys, in the liver, in the capsule of the spleen, and in other organs, which leads to the conclusion that it is a constitutional disease.

3. That the frequent occurrence of these pathological changes in persons of a certain build, might lead to the conclusion that they are due to a particular diathesis; and while excessive spirit drinking might probably excite the development of fibroid disease in any diathesis, yet from the fact that these fibroid transformations are generally, if not always, associated with a certain conformation of the body, and that such changes do occur quite independently of the influence of alcohol, it would appear that there is a special fibroid diathesis.

4. That there are certain features of the body by which, in a great many cases, this fibroid diathesis may be diagnosed.

5. That there is evidence to show that such fibroid changes are not due to a so-called scrofulous constitution.

6. That there is no proof that the yellow, "cheesy looking matter," seen in fibroid lungs, has not the same origin as the cheesy looking matter which is seen in fibroid and other new formations elsewhere.

7. That signs of degeneration of the elastic tissues and atrophy of the lung, as indicated by long-standing vesicular emphysema and bronchitis, precede this form of fibroid degeneration; and although by the physical signs alone it is very difficult to diagnose this disease from tubercular phthisis, yet there are features of the body and other evidence by which, in the majority of the cases, such a diagnosis may be made during life.

8. That considering the build of the patients, their hereditary tendencies, the general symptoms and habits of those who suffer from fibroid degeneration, and how different they are from what is usually found in cases of tubercular or scrofulous phthisis, we ought to draw a distinction between this fibroid transformation or fibroid phthisis and the tubercular or scrofulous phthisis?

Lastly, I would acknowledge my many and great obligations to the Medical Staff of Guy's Hospital, and especially to Dr. Wilks, to whom I am indebted for the opportunity of seeing the post-mortem examinations, for many post-mortem records, and, above all, for a great amount of valuable pathological information, which has been always most generously and readily given. I am also indebted to Dr. Barlow, Mr. Durham, and Dr. Hughlings Jackson, for great assistance, and last, but not least, to Mr. Richard Beck, through whose kindness I have been enabled to have these microscopical sketches made.

Appendix.

In the preceding paper I have simply given a record of what has come under my own notice, fully believing that the description of the morbid anatomy of the disease would be at once recognised. Although the disease has not been known under the name of "fibroid degeneration," the pathological changes have long since attracted attention, and have been variously described under different names.

Broussais, Laennec, Chomel, and Andral, have described various indurations of the lung which they have considered to be the result of chronic pneumonia.

Andral, in his 'Clinique Médicale,' especially mentions two kinds of induration—the red and the gray—and, while speaking of the gray induration, remarks that although the colour may vary in different shades, from gray to almost black, yet that all these forms of induration are due to chronic pneumonia. The late Dr. Addison, in his excellent paper on "Pneumonia" (see 'Guy's Hospital Reports,' second series, vol. i, p. 365), describes the gray induration, and a subdivision of it, the granular induration; and at the same time states that he is unable to understand how it is that gray or black induration results from chronic pneumonia. He regards it rather as the result of an acute inflammation, resembling the inflammation of serous tissue. Hasse ('Pathological Anatomy,' Sydenham Society's translation, pp. 227 and 228), speaking of chronic pneumonia, says it scarcely ever arises out of ordinary acute pneumonia, but coincides most frequently with the formation of tubercle; he further remarks that Andral and Hope describe chronic pneumonia as a slowly developed hypertrophy of the septa of the lobules and cells, attended with a gradual deposition of albuminous matter in the interstices of the pulmonary substance. Grisolle, in his well-known work on pneumonia, says (vide 'Pneumonia,' Grisolle, p. 69, 2nd edition), "Pathologists have

given the name of chronic pneumonia to different anatomical forms of induration." Grisolle then refers to M. Charcot's monograph on 'Pneumonie Chronique,' and states that M. Charcot has arranged the different forms of chronic pneumonia under two heads (vide M. Charcot, 'Pneumonie Chronique: Thèse du concours d'aggregation,' Paris, 1860; also, Grisolle, 'Pneumonie,' p. 69, 2nd ed.):—1st. The pneumonie chronique simple—in which the induration attacks the lower lobes—an exceedingly rare form. 2nd. The induration often observed around accidental productions of the lung, and in particular in the neighbourhood of crude tubercles and cavities—a form which he considers very common. Nevertheless, it has always been difficult to decide what chronic pneumonia really is; so much so that, as Grisolle remarks, it is not possible to say what the disease is in the beginning. Dr. Addison seems to have considered gray induration the effect of acute inflammation; and I have several times heard Dr. Wilks, in his excellent pathological demonstrations, observe that Dr. Addison was in the habit of referring to particular cases in which there was a history of acute inflammation of the lung, and in which after death gray induration was found to exist. Nevertheless, as Dr. Wilks stated, he could bring forward cases in which there was gray induration and no such history; and in his opinion the gray induration or chronic pneumonia is a process more allied to a growth than to inflammation, and is essentially chronic from the beginning (vide "Cirrhosis of the Lung," Dr. Wilks, 'Pathological Transactions,' vol. viii, p. 42).

Rokitansky considers these indurations under the head of "Interstitial Pneumonia" ('Pathological Anatomy,' Sydenham Society's translation, vol. iv, p. 82).

On referring to M. Charcot's monograph, and especially to the description of the microscopic examination of the lung in so-called chronic pneumonia, both by himself, by M. Charles Robin, and by Professor Heschl, of Cracow, it will be probably concluded that, although our descriptions do not agree verbally, yet that they refer to tissue morphologically the same. I

have no doubt that the morbid appearances described as fibroid degeneration of the lung are such as have been not unfrequently considered under the names of chronic pneumonia or gray induration; but I would especially beg to mention that it is not proposed to class under the head of fibroid degeneration all cases in which there is more or less fibroid induration of the lungs. Thus, the small circumscribed fibroid masses which Dr. Addison regarded as the result of lobular pneumonia; the same sort of masses supposed to be referable to pyæmia cured (see 'Guy's Hospital Post-mortem Records'); the induration resulting from the curative process around tubercular phthisical cavities; the induration in the lungs of the millstone grinders; the miner's phthisis described by Dr. Peacock, and the fibroid nodules of syphilis, do not come within the meaning of the term, which it is proposed to limit to cases characterised by the pathological changes above described—cases in which the connective tissue of the lung is the primary seat of the disease, and in which there is a definite clinical history of vesicular emphysema and bronchitis, with or without a history of acute inflammation of the lungs. We mention this more especially, because we have seen several cases on the post-mortem table at Guy's Hospital, in which there was fibroid induration of certain parts of the lung, and in other parts, circumscribed masses of new tissue composed of a number of imperfectly developed cells such as would be generally regarded as tubercular material, and in these cases, there was well-marked ulceration of the bowels with tubercle in the intestines. Such cases appear to be mixed cases, the bridge of union between true scrofulous and fibroid phthisis.

The class of case which it is proposed to call fibroid degeneration of the lungs has not been overlooked—examples may be seen in Bayle's 'Recherches sur la Phthisie Pulmonaire,' p. 211, also under the head of 'Phthisie avec Melanose,' p. 220; and there are excellent illustrations in Andral's 'Clinique Medicale,' translated by Dr. Spillan, p. 305, and in Stokes' 'Diseases of the Chest,' under the head of 'Tubercle the Consequence of a Chronic Bronchitis,' page

426, and in Dr. Laycock's 'Remarks on Rheumatic Phthisis' in the 'Med. Times and Gazette,' 1862, vol. i, p. 451.

There is one more question which it is necessary to determine, viz., whether we ought to consider the cirrhosis of the lung described by Corrigan as identical with fibroid degeneration of the lung?

An admirable case of the former disease was brought under the notice of the Pathological Society, during last session, by Dr. Barlow; and the microscopical examination of the diseased lung showed the induration to be due to fibroid tissue. In a case of cirrhosis of the lung under the care of Dr. Addison, one lung had the appearance seen in cirrhosis, that is to say, the entire right lung was converted into a tough fibroid tissue, while in the opposite lung there were some indurated masses resembling what is seen in fibroid degeneration. Similar appearances were also seen in a case of cirrhosis of the lung under Dr. Gull (see 'Guy's Hospital Post-mortem Records,' 1865, No. 83, in Dr. Addison's case, No. 203, 1865, and in a case of Dr. Hughes, No. 98, 1859.

Why in a man given to intemperance one lung should be affected with fibroid induration and not the other, is no more difficult to understand than why at times in the body of a drunkard we should find the fibroid induration limited to the capsule of the spleen or liver, or the connective tissue of the liver only affected. The determining cause of the local change is beyond our knowledge; but we every now and then see cases in which the same morbid condition of the lung is associated with similar fibroid changes in other organs, the general cause being in all the same—alcohol. Thus the conclusion is forced on us, that the mere localization of the disease is dependent upon some cause apparently accidental, but that in each case there is the same impaired nutrition of the fibrous tissues of the body, and in consequence the same general or constitutional condition of the patient. Bearing the above facts in mind, it appears to me that cirrhosis of the lung may be properly classed under the general term of fibroid degeneration of the lung,

and that there is not in cirrhosis any particular morphological change peculiar to that disease alone. In fact I would endorse the remarks of Dr. Wilks, to whose learning and experience I never can sufficiently express my obligation, that cirrhosis is merely the extreme development of a morbid condition such as is every day seen in a less degree in phthisical lungs.

In the following table, which is principally compiled from the 'Guy's Hospital Post-mortem Records,' are given the particulars of thirty-four cases in which fibroid degeneration of the lung was discovered after death.

	No. of Post-mortem Record.	Year.	Initials.	Age.	Sex.	Under the care of	Occupation.	Habits.	Previous ailments and condition on admission.
1	118	1857	—	57	M.	Dr. Hughes	—	Intemperate	Had suffered with cough and shortness of breath for eight years
2	83	1856	—	56	M.	Mr. Cock	—	Intemperate	Admitted for fractured ribs; suffering also on admission from bronchitis. Dulness over the left chest
3	20	1858	C. W.	37	M.	Dr. Habershom	—	Not stated	Had suffered with bronchitis
4	226	—	G. D.	37	M.	Dr. Barlow	—	No distinct history of intemperance	Body dropsical. Bright disease
5	5	1859	H. B.	34	M.	Dr. Rees	—	—	Phthisical symptoms for more than two years; œdema of the feet
6	46	—	J. B.	42	M.	Dr. Gull	—	—	Ill some years

Lungs.	Heart.	Liver.	Kidneys.	Intestines.	Remarks.
Right pleura contained old and recent lymph; bronchial tubes somewhat thickened and dilated; upper lobes of both lungs consolidated by iron-gray induration; in the rest of the lungs some hard masses, granular on their surface, thus resembling tubercles; lower lobes in a state of red hepatization	Right ventricle dilated, hypertrophy	Healthy	Granular	Healthy	Urine albuminous
In the bronchi evidence of old disease; the longitudinal fibres very distinct; left pleura adherent; right pleura contained recent lymph and serum; left lung compressed; upper lobe of right lung in a state of chronic pneumonia; many small tubes seen passing through the consolidated portion	Right ventricle dilated, hypertrophy	Healthy	Very much degenerated	Healthy	—
In the lungs large masses of indurated tissue almost black in parts; the masses were granular, thus like to tubercle; no miliary tubercle in the healthy portion of the lungs	Right ventricle dilated, hypertrophy	Extreme cirrhosis	Healthy	Not stated	—
Pleura very thick and tough; left upper lobe consolidated by hard black material; small cavity; right upper lobe consolidated in parts, red hepatization in other parts	Old pericarditis, heart's cavities enlarged	Healthy	Small, contracted	Not stated	Urine albuminous
Pleuræ both adherent; upper half of right lung hard and black; left much the same in appearance, and contained a cavity; tubes dilated; recent inflammation also in right lung	Dilated, hypertrophy right side	Healthy	Large, otherwise healthy	Healthy; no tubercle	—
Left pleura very thick; both lungs consolidated by dark tissue, the result of chronic pneumonia, which in parts was granular, giving rise to an appearance like miliary tubercles, but caused probably only by the contracting inflammatory material; minute tubes dilated	Dilated, hypertrophy right side	Healthy	Healthy	Healthy; no tubercle in any part of the body	—

	No. of Post-mortem Record.	Year.	Initials.	Age.	Sex.	Under the care of	Occupation.	Habits.	Previous ailments and condition on admission.
7	252	1858	M.A.A.	48	F.	Dr. Gull	—	—	Jaundice, &c.
8	59	1859	J. S.	30	M.	Dr. Rees	—	—	Fatal hæmoptysis
9	76	1858	T. M.	38	M.	Dr. Hughes	—	—	Bronchitis and emphysema
10	98	—	G. N.	37	M.	Dr. Hughes	Policeman	—	Died of tumour of the brain
11	203	1859	G. P.	62	M.	Dr. Addison	Engine driver	—	Ailing four or five years; admitted for bronchitis; right side dull; systolic bruit at right apex; legs dropsical
12	42	1861	J. D.	50	M.	Dr. Wilks	—	—	Bronchitis for years
13	43	—	P. B	56	M.	Dr. Pavy	—	Free liver	Paralysis left side; softening of the brain

Lungs.	Heart.	Liver.	Kidneys.	Intestines.	Remarks.
Chronic pneumonia of the upper lobes	Healthy	Cirrhosis	Healthy	—	—
Lungs quite healthy, except at the apices, which were indurated by masses of chronic pneumonia; one of these masses had softened, formed a cavity opening into which was a bronchial tube and a branch of pulmonary vein; no tubercles in any part of the body	—	Healthy	Healthy	Healthy	—
Lungs in parts markedly emphysematous; both upper lobes consolidated; hard black masses; in other parts the indurated masses were granular, such as might be called miliary tubercle; no isolated tubercle elsewhere	Dilated, hypertrophy right side	Nutmeg	Healthy	Healthy	—
Left pleura half an inch thick; right, upper, and middle lobes in a state of cirrhosis; opposite lung contained small masses of chronic pneumonia; granular like to tubercle	Healthy	Small, containing earthy matter	Healthy	Healthy	—
Right pleura adherent; right lung cirrhosed from end to end; a tough fibroid tissue; hardening process seemed to have commenced in several distinct portions at the same time; colour nearly black; tubes, upper part thickened and rather large, otherwise not much affected; a small cavity at the apex; left lung, some indurated masses showing the commencement of a similar disease; some white, others a black colour	Right side dilated, hypertrophy	Nutmeg	Large and granular	Healthy	—
In the upper lobes were the hard black tissue of chronic pneumonia, and cavities containing some yellow matter; the lower lobes were in a state of red hepatization	Healthy	Healthy	Healthy	Healthy	—
Lungs in one or two parts contained patches of chronic pneumonia; signs of recent inflammation in the lower lobes	Muscular columns had undergone fibrine degeneration	Cirrhosis	Healthy	Not given	—

	No. of Post-mortem Record.	Year.	Initials.	Age.	Sex.	Under the care of	Occupation.	Habits.	Previous ailments and condition on admission.
14	224	—	T. A.	45	M.	Mr. Birkett	—	Intemperate	Fractured leg; delirium tremens
15	94	1862	W. B.	42	M.	Dr. Barlow	—	—	Chronic bronchitis
16	170	—	H. J.	50	—	Dr. Rees	—	—	Had suffered a long time with chronic bronchitis
17	200	1863	R. L.	63	—	Dr. Rees	—	—	—
18	18	1864	G. F.	38	M.	Dr. Barlow	—	Intemperate	Bronchitis
19	77	—	T. P.	34	M.	Dr. Wilks	—	Very intemperate	Bronchitis for two winters
20	208	—	—	71	M.	Dr. Wilks	—	Intemperate	Suffered with winter cough for years; physical signs of bronchitis
21	209	—	—	50	M.	Mr. Hilton	—	Intemperate	Admitted for injury to the right orbit
22	210	—	—	50	M.	Dr. Rees	—	Drank very hard of spirits	Symptoms of contracted pylorus
23	213	—	—	54	M.	Dr. Gull	—	Intemperate	Cough and dyspnœa for some time

Lungs.	Heart.	Liver.	Kidneys.	Intestines.	Remarks.
Lungs contained a large mass of indurated tissue, the result of chronic pneumonia; tissue solid and of black colour in both lungs and in all the lobes; no evidence of tubercle	Endocardium of the left ventricle thickened and opaque: is it due to "alcohol?" muscle fatty	Very fatty	Pale and soft, probably fatty	Not given	—
Right lung, old adhesions, its structure indurated in various parts from chronic pneumonia	Dilated, hypertrophy right and left ventricle	—	Firm	—	—
Acute pleurisy right side; bronchial tubes full of mucus at their terminations; patches of indurated tissue scattered in parts of the lungs; surface thickened	Acute pericarditis, dilation and hypertrophy of right ventricle, dilated left ventricle with fatty muscle	Nutmeg	Healthy	—	—
Large gangrenous cavity in upper lobe of left lung, also some old consolidation in this and in the other lung	Healthy	Cirrhosis	Healthy	—	—
Portions of the lungs indurated by hard, black, fibroid masses, especially in the upper lobes; in parts had the granular arrangement	—	Cirrhosis	Healthy	Healthy	—
Both lungs much indurated by hard, firm, blackish-gray material; in parts cheesy looking	—	Cirrhosis	Granular	Healthy	—
Hard, firm, and black indurated masses in the upper parts of both and especially of the left lung; masses in lower lobes also; cavities at the apices	—	Fatty	Granular	Healthy	—
Both upper lobes much indurated; granular form well marked; small masses in other parts; lower lobes healthy	—	Cirrhosis	Granular	Healthy	—
Lungs, the upper halves indurated by firm, grayish, hard material like chronic pneumonia	—	—	Healthy	Healthy	—
Upper lobes hard; dark solid tissue; some cheesy matter; a cavity	—	Cirrhosis	Granular	Healthy	—

	No. of Post-mortem Record.	Year.	Initials.	Age.	Sex.	Under the care of	Occupation.	Habits.	Previous ailments and condition on admission.
24	273	—	—	56	M.	Dr. Gull	—	Intemperate	Had a winter cough for years; admitted for Bright's disease; dropsy
25	308	—	—	34	M.	Dr. Barlow	—	Intemperate	Cough for years
26	199	—	—	45	M.	Dr. Rees	—	Intemperate	Had had winter cough and dyspnœa for years
27	197	1865	J.A.N.	51	M.	Dr. Rees	—	—	Brought in for paralysis of the right side; apoplexy
28	86	—	C. L.	58	M.	Mr. Poland	—	—	Admitted in almost a dying state, with disorganization of the knee-joint; chronic cough
29	85	—	F. H.	54	M.	Mr. Poland	—	Intemperate	Fractured leg; acute and chronic symptoms of lung disease
30	83	—	H. G.	29	M.	Dr. Gull	—	Intemperate	Cirrhosis of the lung

Lungs.	Heart.	Liver.	Kidneys.	Intestines.	Remarks.
Left lung contracted to half its size; both very hard when cut into; upper half of both converted into tough hard fibroid tissue; in parts this assumed the granular form; cheesy spots; a cavity at the left apex	Dilated, hypertrophy right ventricle	Nutmeg	Cysts and granular	Healthy	—
Cirrhosis of the left lung; right hypertrophied	Dilated, hypertrophy right ventricle	Nutmeg	Cysts, granular	Healthy	A tricuspid bruit
Emphysema in various parts; chronic pneumonia in upper part of both lungs; portions of the lungs consolidated by dark granular masses; cavity at the apex and small cavities; dilated tubes	—	Healthy	Healthy	Healthy	—
Old and recent disease of the lungs; the upper lobes contained much hard black tissue; this in one or two places apparently softening into small cavities; rest of the lungs in a state of red hepatization	Left ventricle hypertrophied	Fatty	Granular	Healthy	—
Chronic pneumonia of various parts of the lungs; masses of indurated tissue, of dark fibroid appearance; some softened into small cavities	—	Healthy	Granular	Healthy	—
Acute pleurisy right side; signs of old and recent bronchitis; minute tubes dilated, though both lungs had masses of chronic pneumonia; in parts granular and tubercle like	Fibroid change in the mitral and muscular tissue	Healthy	Granular	Healthy	—
Left pleura, over the upper lobe, about half an inch thick; in the upper lobes of the right lung small masses of black indurated tissue, and on the right pleura some soft lymph, but no marked thickening of the pleura; left lung indurated throughout, except at the extreme base, by hard, dark material	Right ventricle dilated, hypertrophy	Much thickened. Capsule very much so, and contracted	Granular	Healthy	—

	No. of Post-mortem Record.	Year.	Initials.	Age.	Sex.	Under the care of	Occupation.	Habits.	Previous ailments and condition on admission.
31	201	—	—	80	M.	Mr. Cock	—	Always been temperate	Stricture of the urethra
32	75	—	—	65	M.	Mr. Durham	—	No evidence	Admitted for hernia; old standing bronchitis
33	Private patient	—	—	40	M.	Dr. Sutton	—	Intemperate	Bronchitis and emphysema
34	Out patient at the Metropolitan Free Hospital	—	—	45	M.	Dr. Sutton	—	Very intemperate	Winter cough for four years; had had profuse hæmoptysis

Lungs.	Heart.	Liver.	Kidneys.	Intestines.	Remarks.
Both apices indurated to the extent of about two inches in depth by almost black tissue, and in the apex a small cavity; most of the disease was seen in the connective tissue of the two upper thirds of both lungs, which was greatly thickened, and in many parts the thickened tissue was putting on a granular appearance like to miliary tubercle; nowhere any granules except amongst the increased connective tissue; the bases of the lungs appeared healthy	—	—	Suppurative nephritis	No ulceration anywhere	There was a small ulcer situated in the larynx
Connective tissue of the upper lobe much thickened, giving a filamentous appearance to the lungs	—	—	Granular	Healthy	—
Pleuræ adherent; bronchial tubes contained much purulent matter; lining membrane very vascular; hard blackish-gray masses scattered through the upper portions of both lungs	—	Cirrhosis	Granular	—	—
Pleuræ adherent; minute bronchial tubes dilated; superior part of the upper lobes of both lungs consolidated with hard gray material in part almost black; degenerating in parts and beginning to turn yellow; a few small cavities like dilated tubes; in right apex a large ragged cavity.	—	Very firm	Large and firm, early granular condition	Healthy	—

DESCRIPTION OF PLATES VII—X.

PLATE VII.

FIG. 1.—A. The corpuscles and cells seen in fibroid lung.
B. The granular appearance. Showing the granules are the contents of delicate fibres. The margins of the new fibres are in this engraving too distinct.

FIG. 2.—Showing cellular elements lying everywhere amidst the elastic fibres of the air sacs, and proving that the pulmonary cells are not simply filled up by the new tissue.

FIG. 3.—Showing the air-cell completely occluded by the new fibroid tissue, and the linear markings of the fine spindle-shaped fibres.

FIG. 4.—Fibroid lung.
Showing spherical-shaped connective tissue cells.
„ granular matter.
„ elastic tissue of the air-cell.
„ advanced stage of fibre-like change; and a number of spindle- and other shaped cells here and there scattered amidst the fibre-like tissue.

FIG. 5.—Spherical and spindle-shaped cells lying amidst the newly formed fibre-like tissue.
Drawn under the 1-20th of an inch object-glass of Messrs. Smith and Beck.

PLATE VIII.

FIG. 1.—The normal elastic fibres of the lung. This drawing is inserted here in order that the elastic may be contrasted with the newly formed fibres.

FIG. 2.—Bundles of newly formed connective tissue. Some of the elastic fibre of the lung seen running across.

FIG. 3.—Section of the granular body—showing a number of corpuscles lying amongst a delicate fibre-like tissue.

FIG. 4.—This drawing was made from the same section as that marked Fig. 3. It shows the corpuscles and fibre-like tissue still more highly developed than those seen in Fig. 3, and the corpuscles are gradually lost as the new fibres are more distinct.

FIG. 5.—This drawing was also taken from the same section as Figs. 3 and 4. It shows a fibre-like tissue crossed by the elastic fibres of the lung; the corpuscles gradually fading and lost in the more highly developed new tissue. This portion of the section included some of the thickened interlobular tissue.

Fig: 1.

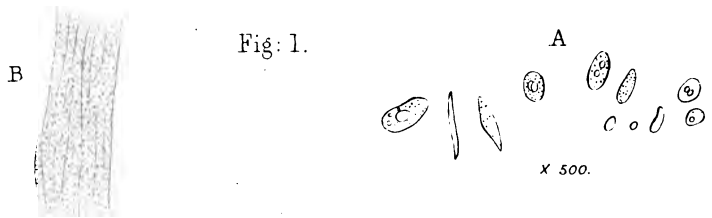


Fig: 2.

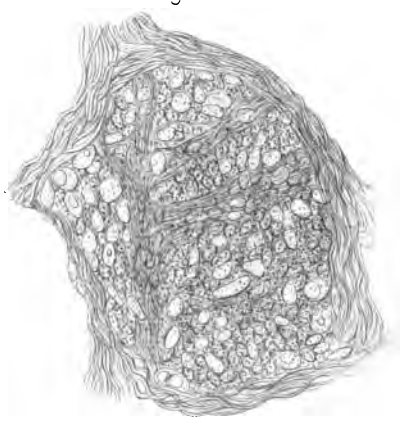


Fig: 3.

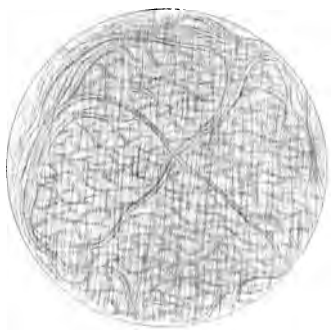


Fig: 4.

Fig: 5.

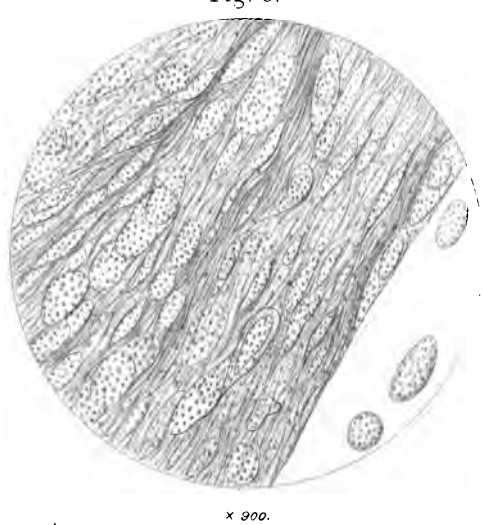
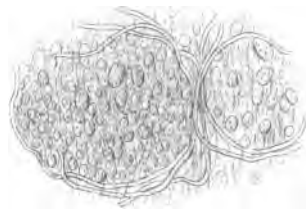


Fig: 1.

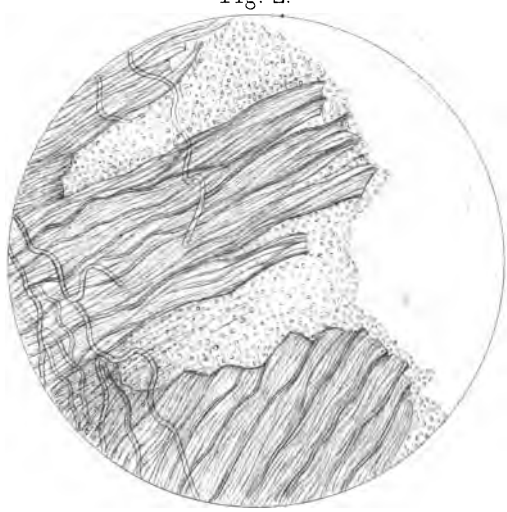
× 750.

Fig 3.



× 400.

Fig: 2.



× 870.

Fig: 5.

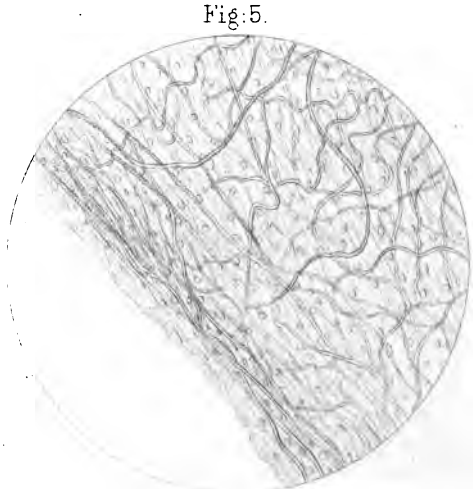


Fig: 4.

× 400.

A.T. Hollick, del. G.H. Ford. × 400. W. West, imp.

Plate IX.

Fig. 1.—Fibroid lung; showing the connective tissue of the lung much increased; the increase in the quantity of pigment and the polygonal spaces.

Fig. 2.—Ditto; showing apex of the left lung. Pleura very much thickened. The lung substance solid and firm; all trace of healthy lung destroyed by the contraction of the fibroid material.

Fig. 3.—Ditto. A portion of the upper lobe of the right lung of the same patient. It shows the blueish-black new fibroid tissue—the granular form—extending into the healthy lung substance. The appearance is such as might be at first sight considered to be owing to the presence of miliary tubercle; but, as described in the paper, the granular appearance is produced by the contraction of the fibroid tissue.

Plate X.

Fibroid lung; the upper two thirds completely solid; the appearance here given is that of a good example of the advanced stage of fibroid degeneration and induration. The pleura is considerably thickened.

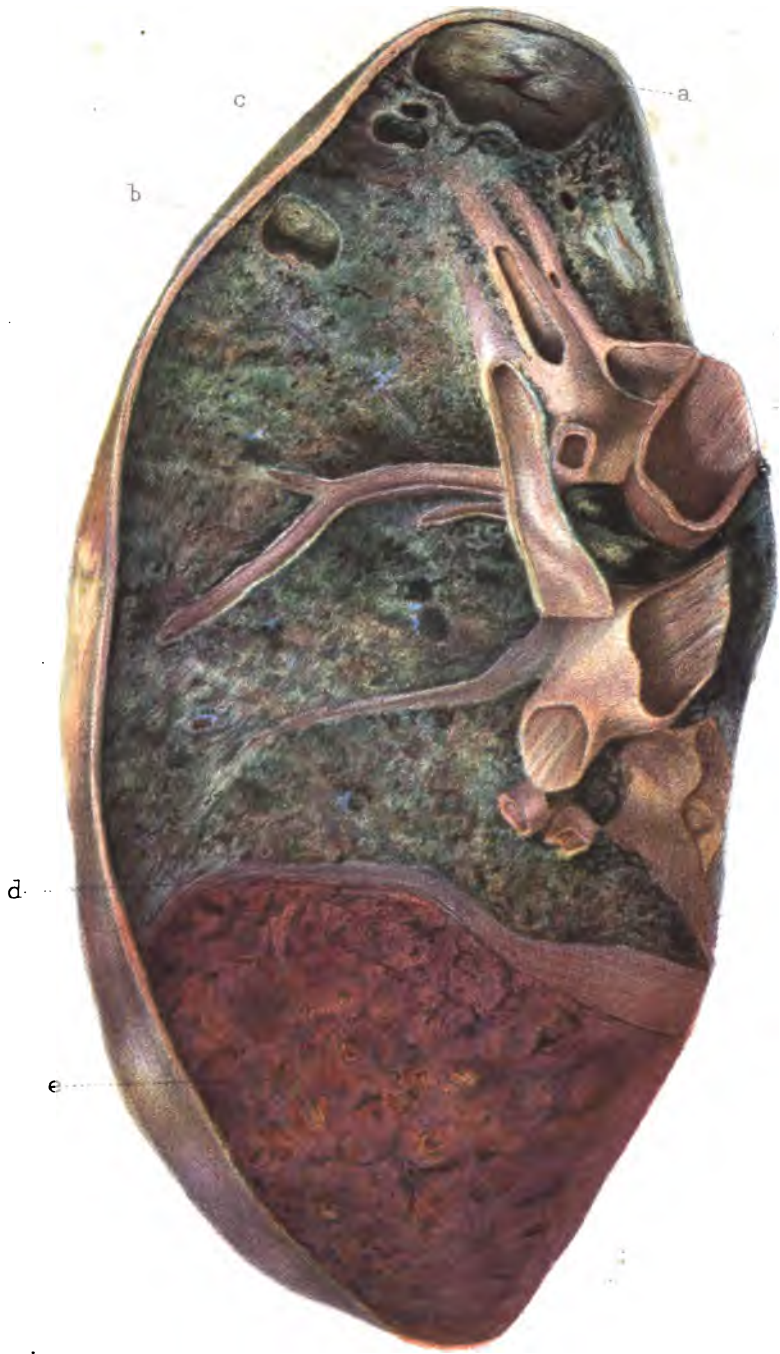
a. An irregular cavity.
b. A dilated bronchial tube.
c. Thickened pleura.
d. Fibroid septum extending from the thickened pleura into the lung substance.
e. Healthy lung.

Fig 1.

Fig 2.

Fig 3.

W.Hurst del. G.H.Ford chr. W.West Chromo Lith.



W. Hurst del. G.H. Ford chr.

W.West Chromo lith.

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